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THE UNIVERSITY OF ALBERTA

THERAPEUTIC APPLICATION OF EEG ALPHA BIOFEEDBACK IN ESSENTIAL HYPERTENSION AND CARDIAC ARRHYTHMIAS

by

EDWARD LYLE GROSS (C)

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH

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FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled "Therapeutic Application of EEG Alpha Biofeedback in Essential Hypertension and Cardiac Arrhythmias" submitted by Edward Lyle Gross in partial fulfilment of the requirements for the degree of Master of Science.

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ABSTRACT

The present study was designed to test the therapeutic effectiveness of EEG alpha biofeedback. Three hypertensives and three arrhythmia patients were referred to the study by their respective physicians.

One baseline session was run before alpha conditioning. Alpha feedback training ran for 13 individual sessions (two per week) for the hypertensive and arrhythmia groups. There were 11 feedback sessions (two per week) for the control group.

Each session was divided into three periods. Period 1: eyes open, prefeedback recording ran for two minutes; Period 2: eyes closed, alpha feedback ran for 15 minutes; Period 3: eyes open, postfeedback recording ran for two minutes. Heart rate, pulse pressure volume, and blood pressure were continuously monitored during each session.

Results showed a lessening in degree of symptoms for some of the patients either within each session or by the conclusion of the final session. Hypertensives showed statistically significant drops in blood pressure during each feedback period. Two of the three hypertensives also had a significant decrease in blood pressure from baseline to the final session. Two of the three arrhythmia patients showed significant changes in their clinical symptomatology one month after the conclusion of the feedback program.

Percentage alpha activity was found to correlate significantly to mean arterial pressure; for the hypertensives only. No other

correlations between physiological parameters were found to exist.

Motivation was found to be important in determining the overall therapeutic success.

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INTRODUCTION

Biofeedback is a term first used only six years ago, and refers to any technique using instrumentation in which a person receives immediate and continuous information about a specific physiologic process which is under control of the nervous system. Biofeedback studies have encompassed many organ systems including heart rate, blood pressure, blood flow, muscle activity, skin resistance, and of immediate interest, brain wave activity. Self-control of these functions has been shown to be possible through biofeedback techniques (Blanchard, 1974).

As a therapeutic tool, Biofeedback:

> "...adds the missing ingredient to the health care team--the patient himself."

Rather than the continual acceptance of pills and treatment from others, the patient can actively partake in keeping or becoming healthy.

Theoretical Considerations: Biofeedback, Pavlovian and Operant Conditioning

Pavlov's work with dogs and the conditioned - reflex method of controlling the autonomic or involuntary nervous system, has led to the traditional belief that visceral function is beyond the realm of voluntary control. Similarly, Skinarian or Operant conditioning was considered to be an effective tool in shaping behavior which involved somato-motor or voluntary functions. Biofeedback has blurred these distinctions.

Thus, a person receiving visual or auditory feedback, which is

modulated by some physiological process (voluntary or involuntary), can master self-control over that activity (Barber et al., 1972, 1973).

Early Human Operant Conditioning Experiments

Although the concept of biofeedback conditioning per se is relatively recent, operant or instrumental conditioning of autonomic nervous system activity had been attempted about ten years earlier.

Kimmel (1974) reported that initial work in operant conditioning of the autonomic nervous system in humans began around 1960. In the Soviet Union, subjects were trained to control digital vasoconstriction and vasodilatation. Using a shock-escape and avoidance paradigm, plus direct visual feedback indicating changes in vasomotor tone, subjects learned to terminate shocks by means of vasodilatation. Control was possible only when direct and real feedback was made available to the subjects.

Kimmel also reviews a number of articles published, in which the galvanic skin response (GSR) had successfully been conditioned. However the skeletal involvement in GSR conditioning was always in question.

Crider et al. (1966) found 40 percent of his subjects showing greater than .5 mv change in skin conductance when using tonal feedback. Simultaneous recordings of electromyographic activity (EMG), respiration rate, and heart rate showed slight indications that the ectodermal responses might be influenced by these physiological parameters. A mechanism for ectodermal control was subsequently proposed. Under conditions of novelty, operant reinforcement (of ectodermal activity), might result in a modified feedback loop, in which covert responses produced stimuli feeding back into the submotor center, eliciting an

ectodermal response, followed by a reinforcing stimulus.

Birk (1966) attempted to resolve the question of whether control over autonomic activity had been learned; or if the results were simply artifactual, resulting from responses mediated by voluntary muscle activity. His methodology involved partial curarization of the skeletal muscles in one subject. GSR activity was recorded from the right palm, and a reference electrode placed on the right forearm. The subject was reinforced with a 70 db tone lasting 1.5 sec, triggered during a skin deviation of .1 mv in the desired direction. Initial conditioning of six sessions ran over a three week period. Curarization was done 20 days after the sixth session, and during this time the subjects only movement was a slight rolling of the head. Results showed that both drug and non-drug sessions were similar in heightened frequency of responses, when contingent feedback was employed, and that during noncontingent reinforcement period a similar decrease in response was noted. Birk concluded that the autonomic conditioning was a derivative of a behavioral response, and that no correlation existed between ectodermal activity and continuous measurement of somatic activity; irrespective of the incompleteness of curarization.

These early experiments in operant conditioning of autonomic nervous system activity were sketchy, and left many doubts as to the validity of shaping autonomic activity without skeletal involvement.

Operant Conditioning of Animal Autonomic Activity

Work on the salivation reflex in dogs, and heart rate control in curarized rats by Miller (1969), and Trowill and Miller (1967), indicated

more definitively that Pavlovian and instrumental conditioning could not be theoretically separated.

Earlier observations indicated that salivation usually did not occur as a result of water reward, and it was therefore assumed that if a dog could be taught to increase or decrease salivation using water as a reward, this conditioning would not be classified as classical or Pavlovian conditioning. Results indicated that salivation could be conditioned in a statistically significant fashion; however, although no skeletal involvement was noted, the possibility could not be ruled out.

A technique first established by Trowill and Miller (1967) involved heart rate control in completely curarized rats. Rats, while maintained on a respirator, were immobilized with d-tubocurare. Reward was electrical stimulation of the medial forebrain bundle. Criterion levels for reward were established; either an increase or decrease in heart rate. Trowill found small changes in heart rate; approximately 5% in the desired direction. Although the change in rate was small, it indicated that the autonomic nervous system could be conditioned without subsequent interference from the skeletal musculature.

Miller (1967) further studied autonomic conditioning in rats under complete curarization. Using a similar procedure as previously outlined; Miller divided rats into two groups: heart rate increases, and heart rate decreases. Criterion levels were first established at a two percent change, and if after 10 minutes it was not met, the criterion level was set slightly lower. The reward circuit and time in signal were turned on, and when criterion level was attained, reward was turned off. Results for rats rewarded for an increase in heart rate (422 to 500 beats

per minute), and those rewarded for decreasing heart rate (400 to 316 beats per minute), showed a progressive change; approximately 20%.

Impulses from the motor cortex still might mediate the observed changes; therefore in order to rule out the involvement of the motor cortex, shocks were administered to the rats' tails. The largest attainable heart rate change was 10%; verifying the lack of skeletal involvement.

Specificity of learning within the autonomic nervous system, has also been investigated by Miller's (1969) group. Rats were taught differential control over heart rate and intestinal contractions. Under complete curarization, and divided into two groups, intestinal contractions were recorded using a water filled balloon placed approximately 4 cm beyond the anal sphincter. A transducer was used to record pressure changes.

One group was rewarded for changes in the first response, and the other group rewarded for the second response. Results indicated that differential control over autonomic activity could be attained, and learning was specific; therefore, learning was not simply a generalized homeostatic mechanism.

Using similar methods as already outlined; conditioning of brain wave activity in cats had been demonstrated (Miller, 1969). One group being rewarded for low voltage, fast activity; and a second for high voltage, slow activity. Successful self-control over the respective brain rhythm was gained, and behavioral differences could also be identified to each of the two groups.

Miller has extensively covered the area supporting independence

of somato-motor and autonomic-visceral control, and others have verified his findings (DiCara, 1971; Pappas, 1970; Harris, 1971).

From Miller's work, and the work of others, this area of research has evolved into an intense and energetic approach towards instrumental conditioning of the human autonomic system.

DiCara (1971) states that:

> "...these findings, which have profound significance for theories of learning and the biological basis of learning, should lead to better understanding of the causes and cure of psychosomatic disorders and of the mechanisms whereby the body maintains homeostasis or a stable environment."

Current Clinical Biofeedback Studies

From a clinical point of view biofeedback studies have involved a number of organ systems; mostly in the area of psychosomatic disorders.

Cardiovascular System. Conditioning of the cardiovascular system has been the most extensively studied area in biofeedback, and with somewhat disappointing results (Blanchard, 1974).

Benson et al. (1971) conducted blood pressure control training sessions with seven patients suffering from essential hypertension. Blood pressure was monitored by an automated constant cuff-pressure system; using a standard size cuff. A microphone was placed under the cuff, over the brachial artery. The cuff was inflated close to systolic blood pressure and any increase or decrease in systolic pressure; relative to each heart beat, could be ascertained. The cuff was inflated for 50 heart beats, then deflated. Median systolic blood pressure was established to be equal to cuff pressure when 14 to 36 Korotkoff sounds, per 50 beat cycle, were present. If less than 14 Korotkoff sounds were

heard, cuff pressure exceeded systolic blood pressure, and the cuff was decreased by 4 mm Hg for the next cycle. If more than 36 Korotkoff sounds were heard, cuff pressure was lower than systolic blood pressure, and the cuff was increased by 4 mm Hg. Initially 30 trials were run, and blood pressure was determined.

Binary feedback (yes-no) to the subject was in the form of lights and tones. Benson found that although systolic blood pressure did not change significantly during the total sessions, it did decrease an average of 4.8 mm Hg within each conditioning session.

Schwartz and Shapiro (1973) extended this study using seven essential hypertensives. Proportional continuous visual feedback; as to what the blood pressure was, was employed. Some subjects showed a 5 mm Hg decrease in diastolic pressure during the session; however, no overall decreases in diastolic blood pressures were observed.

Schwartz (1972) also examined differentiation and integration of learning within the cardiovascular system; thus trying to substantiate Miller's earlier finding with rats. The term integration referred to any change of two physiological processes occuring in the same direction. Differentiation referred to a response pattern in opposite directions. Schwartz found clear evidence only for differential control of blood pressure (down), and heart rate (up).

Blanchard (1974) cites two studies: One by Miller in which a single subject conditioned his blood pressure to change 10 mm Hg in either direction; and another by Elder, showing a 20% decrease in diastolic blood pressure in 18 subjects; each was conditioned over 8 sessions.

Heart rate control and its application to the cessation of various

forms of cardiac arrhythmias, has warranted a great deal of research effort. The majority of studies are only anecdotal case reports, and therefore are subject to considerable criticism; which Blanchard (1974) covers thoroughly.

Engel (1972) published early reports of the clinical aspects of heart rate control. The basis for his initiating the work is outlined in the following paragraph:

> "By the time we began our experiments, we already had carried out three cardiac conditioning studies with normal subjects, and Miller and his colleagues had shown that it was possible to operantly condition heart rate in the rat. Furthermore, there was extensive literature that showed that the nervous system could play a major role in modulating the prevalence of cardiac arrhythmias, and that showed that temperamental variables affected the prevalence of many arrhythmias. This data indicated that the nervous system exercised a significant role in cardiac function, and at least some aspects of this nervous control were associated with volitional behavior (1973)."

Engel has also reviewed the cardiodynamics, hemodynamics, neural and psychological regulations in operant conditioning of cardiac function. As a result of conditioning heart rate, Engel states that patients have become less anxious about their arrhythmia, and they have attained a sense of mastery over themselves. Although they found it difficult to objectively describe how they learned heart rate control; subjectively, generalized relaxation seems to be of prime importance.

Weiss and Engel (1971) have shown a decrease in premature ventricular contractions (PVC) in five of eight patients treated. Using binary feedback in the form of lights and tones, sessions ran from 22 to 53 in number.

Subsequent studies by Bleecker and Engel (1973) examined the control

of supraventricular tachyarrhythmias. Using a similar technique as described by Weiss and Engel; positive results were obtained from several patients suffering from sinus tachycardia, paroxysmal atrial tachycardia, supraventricular tachycardia, and atrial fibrillation. In describing the predominant comments made by the patients, decreased anxiety about his heart was consistent.

Epidermal Response. Sargent (1973) has examined the alleviation and prevention of migrane and tension headaches using a combination of autogenic phrases and biofeedback. Two parts were formed; one focussing on passive concentration and relaxation of the whole body, and the other group focussing on achieving warmth of the hands. The phrases were memorized before the first session. Feedback conditioning required the use of a temperature trainer. Using thermisters, the differential temperature was determined between the midforehead and the right index finger. Subjects were seen on a weekly or biweekly basis; their task being to concentrate on the learned phrases, as well as increase the temperature of their hand relative to the forehead. In two to four months, sessions were reduced to once monthly for a mean of 7.7 months. Results indicated a 63% improvement in reducing the frequency of migrane attacks, and a 33% improvement for tension headache sufferers.

According to Sargent, the mechanism involved is as follows:

> "The symptoms of a migrane attack are mediated through the autonomic nervous system and are often evidenced by increased blood flow in the head. In muscle contraction headaches, one group of investigations has demonstrated increased blood flow through the affected muscle. Thus, both migrane and tension headaches must be ameliorated by regulation of blood flow through voluntary control of the autonomic nervous system."

Muscular System. In a therapeutic capacity, biofeedback within the muscular system has presented the most encouraging results. Muscle (EMG), feedback has been investigated by a number of authors for the alleviation of tension headaches, and the lowering of chronic anxiety (Raskin, 1973; Budzynski, 1969).

Raskin (1973) presents a study in which EMG therapy was employed as a technique for relaxation training in chronic anxiety states. The study involved 10 subjects; 6 suffering from insomnia, and 4 suffering from tension headaches. Conditioning focussed on a reduction of EMG activity in the frontalis muscles. Five of the six insomnia patients showed improvement; while all 4 tension headache sufferers showed improvement.

Primarily concerned with tension headaches, Budzynski and Stoyva (1969, 1970, 1973) have presented a series of studies in which they applied EMG feedback as a therapeutic tool.

Initially, Budzynski established three goals which biofeedback should fulfill:

1. The development of increased awareness of the relevant physiological function or event.
2. The achievement of control over these functions.
3. The transfer or generalization of that control from the training state to other areas of ones' life.

Six patients with a long incidence of tension headaches were given proportional tonal feedback indicating high or low EMG activity. Training varied in duration from 4 weeks to 2 months, depending upon the

individual's level of headache activity. As training progressed, a reduction in the frequency of tension headaches resulted. By the third and fourth weeks, a significant reduction in headache frequency existed.

Budzynski stressed that the core of the technique is based upon precise measurement, and amplification of a particular response, information feedback and shaping, and this technique could therefore be applicable to a variety of physiological events.

Concurrent studies by Wickramasekera (1972) have shown reductions headache frequency and intensity, and as Blanchard (1974) points out in his review of this article, EMG feedback has been demonstrated to be a potent treatment package for intervention in tension headaches, and that home practice is an important parameter in achieving and maintaining success.

Central Nervous System. Conditioning central nervous system responses (CNS) (in particular brain wave activity), has generated a great deal of enthusiasm amongst researchers and the lay public alike; however, very few clinical studies have been conducted therapeutically applying EEG biofeedback. This is in part due to some of the disappointing data collected concerning the specific brain rhythms (Blanchard, 1974; Travis, 1975).

One area of EEG biofeedback, in which the data seems encouraging, from a therapeutic point of view, has centered around the cessation of epileptic seizures. Work by Sternman (1972) centers around conditioning the sensorimotor rhythm (SMR) which typically occurs from the central region of the cortex. Originally working with cats, electrodes were placed on the dura over the sensorimotor region. It was observed that during the voluntary

suppression of movement, 12-14 cycles per second (cps) activity appeared. It was felt that this activity represented a neural process of motor inhibition. Upon conditioning the rhythm, a motionless posture was maintained. Simultaneous recordings of other physiological activity showed a decrease in tonic motor discharge, while cardiac and respiration rate became more regular. Sleep patterns were also modified by SMR training. Sternman noted that:

> "...sleep spindles were enhanced and the number of motor disturbances during sleep diminished in SMR trained cats."

The overall amount of time spent in sleep was reduced and a most significant finding was that drug induced seizures were resisted.

Progression to human studies involved a similar procedure as that used for conditioning cats. Rewards varied from lights to pictorial feedback. Six patients with epilepsy, and two suffering from spinal end lesions, were studied over a period of 6-20 months. Feedback sessions ran from 20-40 minutes; 3 per week. Findings indicated that the 12-14 cps activity could be controlled when it occurred over the rholandic region of the brain. All subjects showed changes in the distribution of EEG frequencies during training. Normal subjects showed a reduction in occipital and central alpha activity during SMR conditioning. Epileptic patients all showed a decrease in abnormal low frequency discharge patterns, with training. This decrease was progressive and sustained throughout the period of training. Additionally, these subjects showed a slow central cortical alpha pattern (6-8 cps) during performance, and with continued training, theta activity was found to diminish. All epileptics experienced an improvement in their

particular clinical patterns; both grand mal and petit mal manifestations were reduced. The lowest rates of clinical seizure activity in the history of their respective disorders were uniformly achieved within several months of initiation of training.

Subjectively, patients expressed increased general awareness and an improvement in the ability to sustain attention in accordance with the typical reports of a relaxed but alert and focussed state of mind during successful performance.

In an anectodal study, Finley (1973) states that a 13 year old subject was able to decrease the occurance of clinical seizures which occurred on the average of 8 per hour. Over a 3 month period, SMR activity increased by 65% with a subsequent 10 fold reduction in the rate of clinical seizures. The duration of the seizures also showed a decrease.

A common factor in most biofeedback studies has been the subjective state of feeling relaxed. Blanchard (1974) generalizes and says that:

> "...it seems possible that the biofeedback procedures may be only elaborate methods for teaching relaxation."

This may be one of the reasons why such wide spread interest in EEG alpha biofeedback has occurred.

The alpha rhythm (8-13 cps) is typically recorded during the eyes closed, relaxed state (Adrian, 1934; Kamiya, 1970). Subjectively, alpha conditioning has been described as a state of relaxed wakefulness. The subject being mentally calm, yet alert (Kamiya, 1970; Brown, 1972).

Although clinically applying alpha feedback has been proposed by a number of authors (Nideffer, 1972; Miller, 1969; Lawrence, 1972); no extensive clinical studies have been conducted (Blanchard, 1974; Travis,

1975).

The characteristics of the alpha rhythm have been thoroughly scrutinized since its discovery in 1926; yet, little is known about its origin and what the alpha rhythm represents physiologically. It therefore has become increasingly important to examine the whole aspect of the alpha rhythm before applying it therapeutically in biofeedback.

Alpha Rhythm and Biofeedback

Historical Background and Definition. In 1929 Berger showed that electrical activity could be recorded from the surface of the human scalp (Adrian, 1934). Because of his crude equipment, and the fact that electrical activity could not consistently be recorded from the typical research animals used; neurophysiologists tended to take Berger's results lightly. Eventually, Adrian and Mathews (1934) were able to record 10 Hz activity from the optic nerve of the water beetle. When the beetle was exposed to illumination, the 10 Hz activity was extinguished. Thus, the conclusion was drawn that nervous tissue was capable of generating a 10 cps rhythm; as Berger had originally proposed. Abolition of this activity by sensory stimulation also verified Berger's finding that the alpha rhythm, typically recorded during the eyes closed state, could be attenuated upon opening of the eyes.

Eventually, a formal definition describing the alpha rhythm was established by the International Federation for Electroencephalography and Clinical Neurophysiology:

> "...alpha rhythm: rhythm, usually with a frequency 8-13 cps in adults, most prominent in the posterior areas, present most markedly when eyes are closed, and attenuated during attention, especially visual" (Lynch, 1971).

Physiological Origin. Jasper and Andrews (1938) examined the involvement of muscle activity in relation to alpha activity. Basically agreeing with Adrian's assumption, they felt that generation of alpha activity resulted in 10 cps muscle tremor. Simultaneous recordings of finger tremor and brain activity indicated a direct relationship might exist between cortical potentials and the rhythmic characteristics of striate muscle. Visual and auditory stimuli resulted in a suppression of both finger tremor and cortical activity. Closer examination in pathological states (paralysis agitans and petit mal epilepsy) revealed no correlations. Their conclusion was that a relationship existed between cortical and subcortical centers in the normal subject at rest, and this is reflected in the grouping of discharges which reach a final common pathway. The muscle tremor produced in the normal state and the pathological state, result from the same center, and that a change in the firing pattern at the cortical or subcortical level resulted in the tremor observed.

Lippold (1970, 1973) has somewhat modified this hypothesis. He stated that muscle activity generates the alpha rhythm, and in fact alpha activity simply was an artifact of these muscle potentials.

The following factors led to Lippold's proposals:

1. Alpha rhythm and muscle tremor tend to have the same frequency in individuals, and often have a similar wave form; waxing and waning usually occurring at the same time.

2. With increased age, tremor frequency rises in the EMG, delta activity is supplanted by the alpha rhythm.

Waves for tremor frequency against age and alpha frequency against age, coincide.

3. When a person is falling asleep, or at the onset of anaesthesia, tremor and the alpha rhythm tend to disappear at the same time.

4. Abnormal tremor (5 Hz Parkinsonian Component) frequently occurs simultaneously with cortical activity.

Through a number of elaborate experiments; Lippold concluded that alpha results via the following mechanisms:

1. There is a reflex loop from the oculomotor nuclei to the extraocular muscles so that the rhythm recorded from the occiput is generated from these nuclei.

2. Because of the geometry of the skull and orbit, the extraocular muscle potentials are smoothed and appear as waves at the occiput.

3. The tremor of the extraocular muscles may modulate the corneo-retinal potential which again because of skull geometry is recorded at the occiput.

Few authors agree with Lippold's hypothesis. At the present time, the most widely accepted explanation for the origin of the alpha rhythm is that it originates within the thalamus.

Bremer (1935) found that sectioning of the brain stem, between the thalamus and mesencephalon, resulted in no reduction of 10 cps activity occurring from the thalamus and the cortex. He proposed that either the thalamus, the basal ganglia, or the cerebral cortex, were responsible for producing the rhythmic activity. In 1958, after further experiment-

ation, he finally hypothesized that the thalamus was specifically responsible for the cortical spindle activity.

Andersen and Andersson (1968, 1971) extended Bremer's work in locating the origin of the rhythmic activity within the thalamus.

Using anaesthetized decorticate cats; abolition of the anterior third of the thalamus resulted in a noticable change in cortical recordings on the ipsilateral side. Localized destruction of the thalamus, 4 mm lateral to the ventro posteriolateral nuclei complex, produced drastic changes in the cortigram. A longitudinal incision made between the midline and intralaminar nuclei, resulted in no observable changes, contrary to Morison and Dempsy's (1942) midline pacemaker concept for spindle activity.

Cooling of the cortex resulted in a marked reduction in amplitude of spindle activity, but no change in frequency. Reduction of the temperature within the thalamus, resulted in a decrease in discharge frequency, and eventual abolition of spindle activity. Crystalline penicillin was used to affect the cortical spindle activity. When injected into the thalamus, a dramatic reduction in frequency was observed, and according to the authors, this result would be expected if a thalamic triggering device were removed or altered. Penicillin injected specifically into the ventralis posteriomedialis nuclei, and the ventralis lateralis, resulted in slowing of spindle activity.

Andersen and Mason (1971) have since identified three distinct patterns of pacemaker activity. The activity originated from the ventro-basal, dorso-lateral, and medial regions of the thalamus. Lopes Da Sliva (1973) has also verified this finding. Measuring thalamo-

cortico and cortico-cortico coherences, a number of different rhythms could be recorded indicating that several pacemaker sites existed. Interestingly, it was found that cortico-cortico coherences were generally greater than the largest thalamo-cortical coherence. This suggests that the rhythm typically recorded from the surface of the scalp might depend more on intra-hemispherical connections rather than inter or intra thalamic connections.

While the physiological origin of alpha waves requires a great deal more investigation, variables affecting alpha activity have been carefully scrutinized.

Variables Influencing Alpha Occurance. Various researchers have examined a number of general features pertaining to the occurance of alpha activity.

Phillips (1971) has indicated that increased smoking lessens the amount of alpha activity, while London (1970) has shown high amounts of alpha related to high scores of hypnotic susceptability. The most frequent observation has been the attenuation and enhancement of alpha through mental activity (Brown, 1970; Kamiya, 1971; Slatter, 1960).

Slatter investigated EEG correlates of mental imagery. A series of tasks involving mental multiplication, visual imagery, and generalized thinking resulted in the following observations: Active vision and visual recall were associated with blocking or attenuating alpha rhythms; alpha activity was also blocked by nonspecific anxiety.

Glass (1964) found that errors in mental arithmetic were associated with high voltage EEG alpha activity; before the presentation of the task.

De Caire (1970) attempts to categorize populations of alpha producers into three groups: The first group (persistent) has alpha occuring immediately upon closing of the eyes, and sometimes they find it difficult to block the activity. Alpha may even occur during the eyes open state. The second and largest group (responsive) exhibit measurable amounts of alpha activity upon closure of the eyes. The third group (minus) includes only about 5% of the population, and shows no alpha activity, even with the eyes closed.

Although categories have been established according to percentage of baseline alpha; very few behavioral distinctions can be found associated to these three groups. A number of authors have observed that high anxiety states were associated with occurance of low alpha activity (Eskenasy, 1973; Lader, 1966; Branch, 1965). Johnson and Ulett (1959) found small correlations existing between the Taylor Manifest Anxiety Scale (Taylor, 1954) and percentage baseline alpha activity. A high amount of alpha activity represented low anxiety, and conversely, low alpha activity reflected high anxiety states. Costa (1965) also found small correlations existing between percentage alpha and trait anxiety.

Physiological Representatives of Alpha Activity. Galin and Ornstein (1972) have measured and compared left to right EEG power spectrums during verbal tasks, and various spatial tasks. Recording from T_3, T_4, and P_3P_4 (10-20 system of electrode placement), results indicated a ratio decrease for spatial tasks, and an increase in ratio for verbal tasks, i.e. a characteristic pattern of activity existed for these two cognitive modes. Because of the high voltage of EEG alpha activity, it was assumed that alpha contributes a large proportion of the total EEG

power, and therefore the findings suggest that suppression of alpha occurs over the active hemisphere.

Doyle (1974) expanded this study, in which the various frequency bands were examined more closely. Of the five frequency bands, alpha seemed to be the only rhythm that showed significant differential qualities during performance of various cognitive functions. The influence of motor and nonmotor tasks on the EEG spectrum, revealed increases in alpha activity occuring when attention to breathing was performed. Motor tasks all tended to depress ongoing alpha activity.

Butler (1974) and McKee (1974) have subsequently verified these findings.

Mulholland (1965) presents a classical illustration in which alpha production is thought to represent eye movement and eye position. Although his results have been disproven by a number of authors (Fenwick, 1966; Nowlis, 1970; Kamiya, 1969) the suggestion is made that some connection between efferent neural pathways and alpha production exists.

The following, Table 1, illustrates the procedure used by Mulholland; as well as the data accumulated.

Both subjects had an abundant amount of alpha time present in the normal resting eyes closed state, and were thus chosen on this basis. Task I presents data when subjects sat in a dimly lit room and performed the following tasks: eyes open, eyes closed, eyes ahead, and eyes up. Task II found the subjects blindfolded, thereby illuminating any change in visual input as was encountered in Task I. Task III presents data from subjects sitting in a room with intense but diffuse light, whenever alpha was present in the EEG. The fourth task made use of the after-image

TABLE 1

PERCENTAGE ALPHA TIME, RECORDED FROM OCCIPITAL LEADS OF TWO SUBJECTS. ALPHA PERCENTAGE VARIES WITH VARIETY OF TASKS PERFORMED. (1) DIMLY LIT ROOM: (2) SUBJECTS BLINDFOLDED: (3) ROOM FILLED WITH INTENSE BUT DIFFUSE LIGHT: (4) AFTER-IMAGE EFFECT (MULHOLLAND, 1965).

TASK I

	SUBJECT % ALPHA TIME	
	I	II
Eyes Open	7	3
Eyes Closed	84	88
Eyes Ahead	9	4
Eyes Up	86	75

TASK II[a]

	SUBJECT % ALPHA TIME	
	I	II
Eyes Open	53	63
Eyes Closed	79	69
Eyes Ahead	18	50
Eyes Up	82	53

[a]Eyes covered with a blindfold.

TASK III[b]

	SUBJECT % ALPHA TIME	
	I	II
Eyes Ahead	8	41
Eyes Up	9	48

[b]Room filled with intense but diffuse light.

TASK IV[c]

	SUBJECT % ALPHA TIME	
	I	II
Eyes Ahead	13	3
Eyes Up	28	28

[c]Following exposure to bright pattern.

effects in which a bright pattern was projected onto the subject's eyes, and after closure of the eyes, he could visualize the flash for about a minute after initial exposure. Mulholland states that these results clearly indicate that eye position is a significant variable in determining the amount of alpha time. Despite the uncomfortable task of keeping the eyes in an upward position, the alpha occurance was higher than the eyes ahead position. Mulholland concluded that the classic "eyes open - eyes closed" test for alpha may not in fact reflect reduced visual input, as had been supposed, but rather some possible variable as a tendency for the eyes to turn upwards under such conditions.

Lynch (1971) has since outlined two categories that might represent alpha occurance. Alpha may be due to physical or somatic factors; attentional or arousal processes.

Mulholland (1973) has since proposed that alpha occurance is attributed to three visual mechanisms--the "triad of accomodation". The triad is comprised of minimization of the angle between the visual target and the fovea; minimization of target blur and control of the pupils to optimize the level of stimulus input. The occurance of these reflexes blocks the alpha rhythm.

Wertheim (1974) reviews recent evidence indicating that alpha activity is directly associated to oculomotor control. Blocking of alpha activity occurs only when the visual information serves also as a monitoring principle in oculomotor position control.

> "...during highly predictive visual tasks, oculomotor control might become independent of visual information, even when the 'sampling' of this information remains undisturbed. Large periods of uninterrupted alpha activity with eyes open are usually associated with a mental state of general relaxation which, if prolonged

long enough, drift off into a state of drowsiness or sleep."

Wertheim summarizes in saying that two types of eye position control exist; an external (retinal) feedback loop, and some kind of internal control may operate in the absence of retinal information. This internal control may rely on memory functions, probably with respect to prior efferents. The ability to block alpha via mental effort, therefore, depends primarily on past related experiences.

Peper (1971) suggests that the activity which blocks alpha is related to neural efferent activity involved in oculomotor activation. Peper found that blocking of central alpha, resulted from movement responses. This agrees with Kreitman and Shaw's (1965) detection of a small negative correlation between EMG activity and centrally located alpha activity. They found that subjects showed a decrease in visual tracking performance when alpha was present. The blockage of alpha was not due to the onset of light input, but due to oculomotor changes.

In summary, Wertheim makes a general hypothesis: the ongoing occipital alpha rhythm is attenuated, desynchronized or blocked by contamination of the EEG trace by electrical activity evoked during activation of neural mechanisms responsible for attentive oculomotor behavior; while during intentive oculomotor behavior, the occipital alpha trace remains undisturbed.

Heinmann (1971) set forth the proposal that alpha activity may reflect inhibitory brain processes. The ongoing alpha activity represents one's ability to inhibit those sensory inputs which might normally block alpha. The author cites work where sensory deprivation studies have found increasing alpha activity occurring.

An increase in alpha activity (11-12 Hz) was found to occur after the announcement of an electrical shock, to be given a few seconds later. This activity has been described as "preventative inhibition".

Heinmann exposed his subjects to a number of emotional words. EEG and ECG recordings were made simultaneously. A delay in recognition of a word was accompanied by increasing alpha activity. Alpha activity was highest between the time when the information became available, but not reaching conscious awareness, and as the stimulus increased in intensity a stepwise recognition of the word began. Therefore, increases in alpha activity may reflect an increasing ability of the subject to ignore otherwise distracting influences.

Conditioning the alpha rhythm through biofeedback has added to the knowledge about its characteristics of occurance, its correlates and its usefulness as an index of homeostasis.

Alpha Conditioning. Schwartz (1972) has found no relationship existing between EEG alpha conditioning and heart rate control. This indicated that the underlying mechanisms for control were different.

Conditioning alpha has shown some characteristic pattern changes. As individuals become drowsy and pass into sleep, their brain rhythms tend to change from predominantly alpha, to fragmented alpha, to low amplitude theta, and finally delta (Budzynski, 1972). Budzynski also noted a considerable reduction in tonic muscular activity.

Peper (1971) attempted asymmetrical feedback training of parietal - occipital alpha activity. Subjects were instructed to keep either a tone on or off. The tone on was indicative of gross alpha asymmetry between the two hemispheres. Two of the six subjects were able to learn

asymmetrical control. Peper suggests that subjects learn to optimize the conditions under which asymmetry occurred and to autoregulate the unknown processes that mediate the alpha rhythm.

Peper's (1972) examination of localized control involved subjects divided into two groups: Group I was instructed to keep the tone high pitch (high alpha); Group II was instructed to keep the tone low (low alpha). One subject was able to gain significant control. The method of control was through various cognitive tasks, thus substantiating Galin and Ornstein's findings of cognition and EEG alpha occurance.

Hord (1972) recorded spectral activity during self-regulation of alpha. Measuring intensity and coherences between frontal, temporal and occipital leads, revealed little evidence of increases in intensity and frequency ranges other than alpha. Occipital alpha was found to be more coherent with frontal than temporal leads. Slow frontal activity, resulting primarily from eye movements, decreased in intensity during successful alpha regulation.

The most distinguishing feature of alpha remains the subjective description of alpha state during self-regulation.

Initially Kamiya (1969) attempted to see whether the alpha state could be identified. A single subject was told that a bell would ring from time to time, and he was to identify whether or not he was in the alpha state. During the first day of testing the subject was correct 50% of the time. On the second day he was correct 65% of the time, the third day 85%, and by the fourth day 100% of the time--400 successive trials.

This study was extended using 10 subjects. The subjects were instructed to keep a tone on for 5 minutes indicating the presence of

alpha. Within 8 trials, 8 of the 10 subjects were successful. Nowlis and Kamiya (1970) conditioned 21 of 26 subjects to control alpha within 15 minutes. Subjectively, the alpha state was described as relaxing, letting go and not focusing on anything.

Brown (1970) employed the use of colored lights and descriptive words in identifying the different EEG states. Basically the results showed that alpha was associated with pleasant feelings, an increased internal awareness, and tranquility. Many other similar subjective reports have been documented (Lynch, 1971; Peper, 1971; Nideffer, 1973; Budzynski, 1972).

These subjective comments pretaining to the alpha state have generated a great deal of enthusiasm towards alpha biofeedback. However, as Blanchard (1974) recently pointed, no studies have been carried out illustrating the therapeutic value of self-regulation of alpha.

Nideffer (1972) has questioned whether or not EEG alpha biofeedback results in physical relaxation in conjunction with the associated mental state. If this is the case; Nideffer also suggests that it might be possible to therapeutically apply this technique in treating psychosomatic disorders. There would appear to be at least three ways in which subjects might benefit from alpha training.

1. Should the alpha be directly related to physical relaxation, it can be useful in the treatment of hypersensitive conditions and it may increase the subject's ability to withstand stress.
2. If the alpha state reflects lower levels of arousal and a relaxation of normal perceptual and cognitive

control, it may be useful in a variety of ways to increase tolerance.

3. Physical relaxation occuring in conjunction with a state of "mental calm".

Somatic Targets of Psychic Tension

It is well documented in the literature that stress and anxiety are important causes of elevation of blood pressure, occurance of irregular heart rates, tension headaches, and migrane headaches (Branch, 1965; Lader, 1966; Robinson, 1969; Pickering, 1968; Kelly, 1970).

Psychophysiological Correlates. Lader (1966) has presented a series of graphs: Figure 1 indicates the relationship between different arousal levels and the affected physiological parameter.

Physiological Measures of Anxiety. Arousal, as Lader explains, interposes between the physiological measure, and the concomitant emotion. Anxiety may accompany high arousal levels and hence changes in some psychophysiological parameters; although a high index of arousal may not necessarily be an index of anxiety. The reason for this may be twofold. An increase in physiological activity may be secondary to increased physiological needs which might be occurring simultaneously, but independently, and secondly, arousal may cover high effect states other than anxiety. If recording conditions are kept constant, and anxiety is determined via psychological testing, a number of physiological reactions show some specificity to anxiety. Frontalis muscle activity tends to be consistently higher in anxious patients. Blood pressure can be labile or chronically raised during anxious states. Respiration rate is

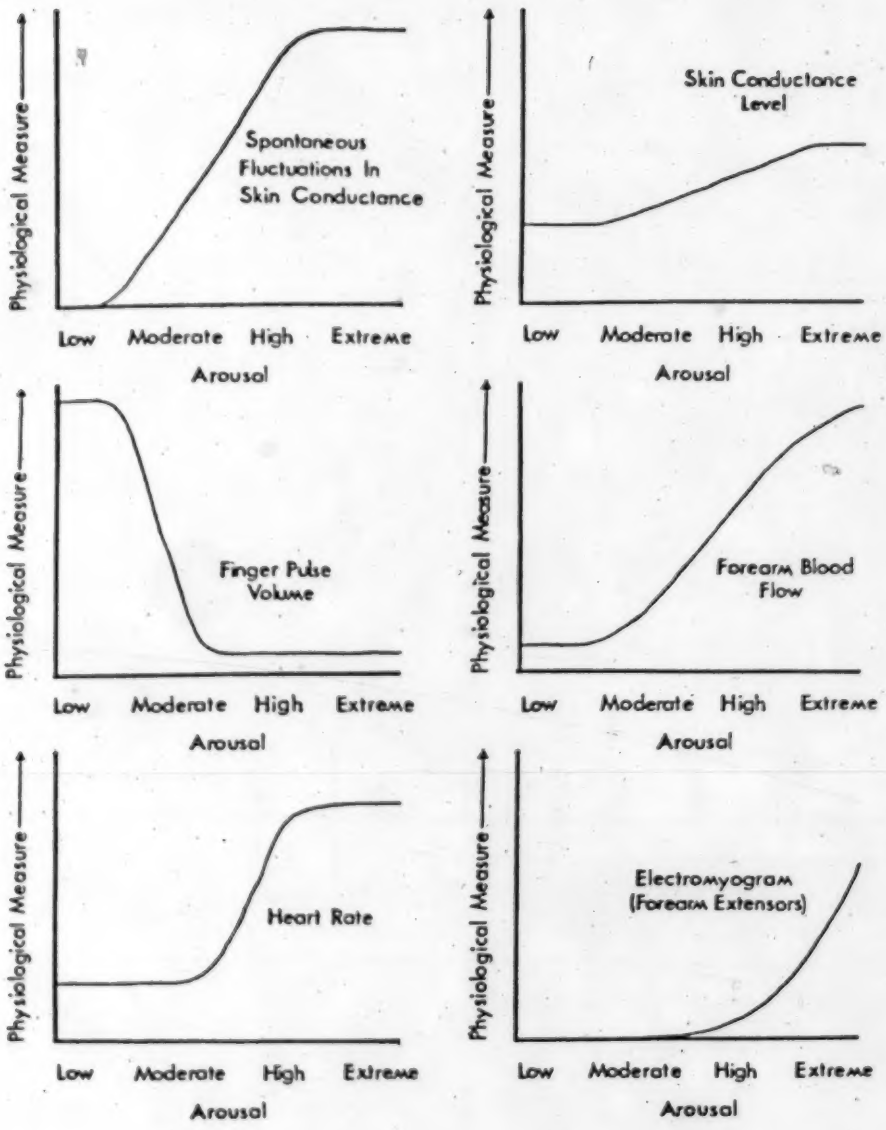

FIGURE 1. Physiological indicators of arousal. This diagram represents the impressions of the author gained from his experience with the measures. The steeper the slope of the curves, the more sensitive is the measure (Lader, 1966).

irregular. 'Intense anxiety' has been associated with vasoconstriction; while 'contentment' with vasodilatation. Faster EEG activity has also been noted during anxiety states, and alpha being less common in chronic anxiety states.

Branch (1965) has extended the influences of anxiety as being responsible for a number of psychosomatic disorders. Anxiety may significantly increase the amount of work required by the heart, inducing arrhythmias--such as paroxysmal atrial tachycardia, premature systoles, atrial fibrillation and paroxysmal ventricular tachycardia. It may cause hypertension or anginal pain may increase.

Pickering (1968) states that environmental factors play a large part in the pathogenesis of the severest cases of essential hypertension and he cites a number of articles demonstrating this.

Robinson (1969) has stated that anxiety may even be a symptom for hypertension (Robinson, 1969). This has been verified to some extent by Vlachakis et al. (Vlachakis, 1974). Patients with labile blood pressure were examined under four conditions. Phase A: Thirty minutes of complete relaxation at room temperature. Phase B: After vasodilatation; produced by heating with electric blankets, a ganglionic blocking agent was infused. Phase C: Conditions of B constant, and infusion of epinephrine chloride. Phase D: Keeping B constant, and infusion of levarterneol bitartrate (norepinephrine base). In all four cases digital blood flow and blood pressures were measured. Results indicated an increased reactivity in the hypertensives. Hypertensives did not score high on "trait" anxiety scales, but did show differences in reactive anxiety. Referring to Malmo, the author states that when an individual with

various psychomatic complaints is subjected to stress, he most likely responds maximally with disturbances in the "critical symptom area"-- i.e. the area in which the psychosomatic complaint was related. It is suggested that anxiety produces more vasoconstriction than increase in cardiac output in the hypertensives, whereas the opposite occurs in the normotensive subject. Wallin et al. (1973) comments that continuous monitoring of blood pressure has shown that many hypertensives react with greater blood pressure elevations than do normotensives when subjected to a variety of stimuli during normal daily activities.

Treatment of Psychosomatic Disorders. A variety of relaxation techniques have recently been used in the treatment of hypertension. Deabler (1973) states that attempting to use psychological methodology in lowering blood pressure is of recent origin. Hypnosis and Jacobson's technique for skeletal muscular relaxation were employed. Twelve patients were divided into two groups: Six for controls; six for experimental use. A 17% lowering of systolic blood pressure was achieved through hypnosis.

Benson (1974) investigated the direct effectiveness of meditation training in hypertensives. Fourteen subjects returned every 2-3 weeks for at least a year after learning the technique. Subjects also remained on their medication throughout the investigation. During the premeditative control period, blood pressure was 145.6 systolic, 91.9 diastolic. During postmeditative experimental periods, blood pressure averaged 135.0 systolic, 87.0 diastolic. Although significant reductions in blood pressure were attained, the long term effects of meditation have not been documented. Benson did notice that his subjects underwent some

behavioral changes during the duration of the experiment.

Redman et al. (1974) found simple verbal instructions effective in eliciting relaxation responses in hypertensives. Over five sessions; predictable directional changes in blood pressure and heart rate occurred in response to simple verbal instruction to alter cardiovascular functions.

Patel (1973) has successfully shown reductions in blood pressure of hypertensives using a combination of yoga and galvanic skin reflex (GSR) feedback. Twenty patients were used for the study. GSR activity was indicated by a tone, an increase in pitch indicating an increase in sympathetic activity, and decrease indicating relaxation. Instructions to the subjects were to concentrate on relaxation. Subjects were seen three times per week for three months, and by this time five patients were able to halt antihypertensive drugs; a 33-60% reduction occurred in seven others. Mean blood pressure dropped from 121 mm to 101 mm Hg. Blood pressure fell by an average of 160 to 134 systolic, and 102 to 86 diastolic. A twelve month follow-up indicated that a statistically significant reduction in blood pressure was maintained. Patel (1975) has also run another group of 20 subjects. Subjects were matched for age and sex, and the results verified the previous findings that blood pressure could effectively be reduced through this technique. A control group was also run, and no reductions in blood pressure were observed. This verified previous assumptions that blood pressure in hypertensives shows no decrease as a result of sitting in a chair in an isolated chamber for a short period of time. Patel postulates that the reduction in blood pressure is brought about by lower sympathetic tone, which is maintained by altered habitual interaction with the environment.

Although alleviation of psychosomatic disorders through various relaxation techniques, including biofeedback, has been well documented, the therapeutic value of EEG alpha biofeedback remains unproven.

METHOD

Subjects

Three male patients with mild to moderate hypertension and three female patients suffering from functional cardiac arrhythmias were referred to the study by their respective physicians.

The hypertensive subjects were 37, 50, and 66 years of age. The arrhythmia patients were 45, 47, and 60 years old. Complete histories of each subject were available for obtaining relevant information and for obtaining accurate baselines. Subjects acted as their own controls. Table 2 tabulates detailed information for each patient.

Six normotensive male volunteers with no history of either reoccurring tachycardia or high blood pressure were used as a separate control group. Ages ranged from 28 to 50 years (S.D. = 9).

Physiological Parameters Monitored

Four physiological parameters were continuously monitored throughout each session.

Heart rate was recorded using silver-silver chloride disc electrodes. Electrode jel (Beckman Instrument, Inc.) was rubbed into the skin removing excess oil. An adhesive disc (polyethylene film; adhesive coated on both sides) was attached to the electrode and the electrode cavity filled with jel (allowing a better electrical contact between electrode and skin surface). One electrode was placed on the ventral side of each

TABLE 2. DETAILS FOR ARRHYTHMIA AND HYPERTENSIVE PATIENTS.

Subject	Sex	Age	Diagnosed Disorder	Duration	Original State Before Commencing Program	Medication
EM	F	45	Supraventricular Tachycardia	3 years	Frequent fluttering lasting approx. 1/2 hour	Digitalis (2 mg) Quinidine (200 mg)
MP	F	47	Paroxysmal Atrial Tachycardia	11 years	Frequent attacks lasting several hours. Can be induced by physical means.	No Medication
AP	F	60	Paroxysmal Atrial Tachycardia	40 years	Frequent fluttering. No serious attack of tachycardia since 1968.	Quinidine (200 mg)
MM	M	37	Essential Hypertension	18 years	Discharged from hospital 2 days before initiating program. BP 134/100.	Hydrodiuril (50 mg)
WL	M	50	Essential Hypertension	13 years	Last recorded BP 1 week before initiating program 180/120.	Apresoline (50 mg) Inderol (50 mg)
EC	M	66	Essential Hypertension	3 years	Discharged from hospital 3 days before initiation of program. BP 143/120.	Apresoline (10 mg) Catapres (2 mg) Aldomet (750 mg)

forearm (Lead I) and a ground electrode was placed on the right wrist.

Pulse pressure volume was monitored using a digital plethysmograph clamped to the right ring finger (Tektronix, Inc.).

Blood pressure was determined using an automated blood pressure system (Avionics Research Products). A microphone was placed under a standard size blood pressure cuff, over the brachial artery of the left arm. A pump automatically inflated the cuff to a predetermined pressure (200 mm Hg). Upon attaining this pressure, the cuff immediately began to deflate. When Korotkoff sounds were detected by the microphone, the pressure in the cuff illuminated the respective pressure found on the face of the instrument (systolic blood pressure). Each falling pressure continued to be illuminated until no Korotkoff sounds were detected. The lower pressure illuminated represented diastolic blood pressure.

The accuracy of this system was compared to a typical sphygmomanometer and found to be 95 percent accurate for both systolic and diastolic pressures. This system was intermittently checked throughout the duration of the program.

Beckman miniature electrodes (11 mm diameter) were used to detect brain wave activity; electrodes attached using double adhesive collars. The scalp was first prepared with alcohol. Electrode jel was rubbed into the skin for approximately 10 seconds. Scalp was wiped clean. The electrode cavity was filled with jel and attached to the skin.

Electrodes were placed (i) 2 cm above the inion (Oz), (ii) on the left ear (A_1), (iii) and on the left mastoid (ground). Oz - ear activity was monitored (monopolar reading). Electrode impedence was kept below 10 kilohms. In order to maintain this impedence throughout the session,

it became necessary for subjects to use the provided headrest, keeping Oz connection stable. This procedure was satisfactory for recording purposes as well as for the subject's comfort.

Instrumentation

Narco Biofeedback System. Brain wave activity was filtered through the Narco Biofeedback System (Narco Medical Services). The system is a solid state modular instrumentation system designed specifically for biofeedback and clinical application. Filters are flat between cutoff frequencies (8 - 13) to $\pm$ 0.5 dB. An adjustable control (from 10 - 200 microvolts) inhibited artifacts before they reached subject's display unit. Proportional audio feedback (i.e. frequency and amplitude modulated with reference to occurance of 8 - 13 (alpha) activity greater than 10 uv) was given via an external speaker situated 8 feet in front of subject. The tone ranged from 10 Hz (10 uv alpha) to 10,000 Hz (>250 uv alpha).

Gated Electronic Timer. 'Criterion alpha' activity (8 - 13 cps) was quantified using an Electronic Timer (designed by Gordon Blinston). This device is an electronic clock with a digital display indicating minutes and tenths of minutes. The displayed value was obtained by counting pulses which occurred at a fixed rate. Counting occurred whenever a signal from the Narco feedback system was applied to the input terminals. Alpha activity greater than 10 microvolts triggered the counting device.

Miscellaneous. All physiological activity, except blood pressure, was recorded on strip chart paper for visual inspection. A 4 channel model 5D Grass polygraph was used. Paper speed was set at 15 mm/sec.

Communication between the subject and experimenter was via an

intercom system.

Periodic visual observation of heart rate, alpha activity, and pulse pressure volume could be done with a Hewlett-Packard oscilloscope connected to the Grass polygraph.

Laboratory Set-Up

The following schematic diagram (Figure 2) illustrates the laboratory set-up. Figures 3 and 4 reflect a photographic view of the testing and experimental chambers respectively.

Experimental Procedure

At the subject's convenience, a time was established at which he would come to the laboratory. This time was kept consistent throughout the duration of the program. Subjects were told that there would be a total of 14 sessions (two per week). Each session would last approximately 45 minutes.

The first (baseline) session consisted of a number of elements:

a. An explanation of the basic relationships between behavioral states (i.e. chronic anxiety, and stress) and physiological activity (heart rate, blood pressure, brain wave activity). In general terms it was explained that underlying stress or anxieties could cause bodily processes to react in a variety of ways: for example, heart rate may quicken or become more noticable, blood pressure may be periodically elevated.

Recent evidence has shown that by producing high amounts of alpha brain wave activity, a subjective state of relaxfulness and mental calm has been reported. What we would like to determine is whether physical relaxation occurs concurrently with the mental state. Thus, we hope to see a lessening in the degree or complete alleviation of the respective disorder.

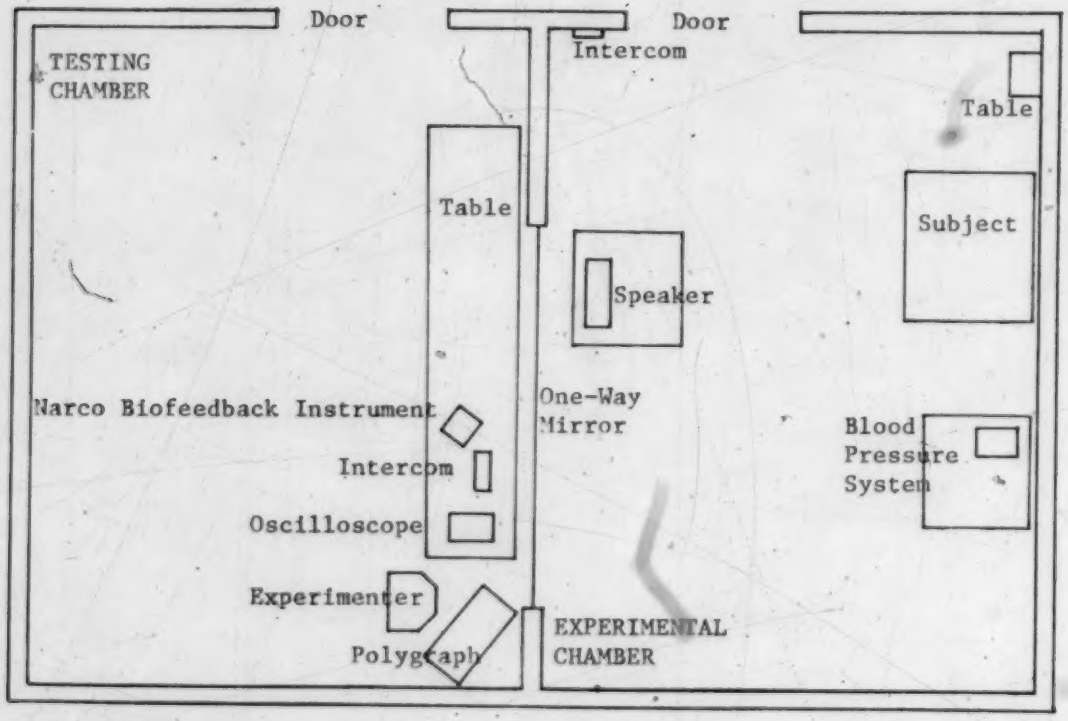

FIGURE 2. Schematic diagram of testing and experimental chambers. Both chambers are 9' X 12'; sound and electrically attenuated (Industrial Acoustics Co., Inc., N.Y.).

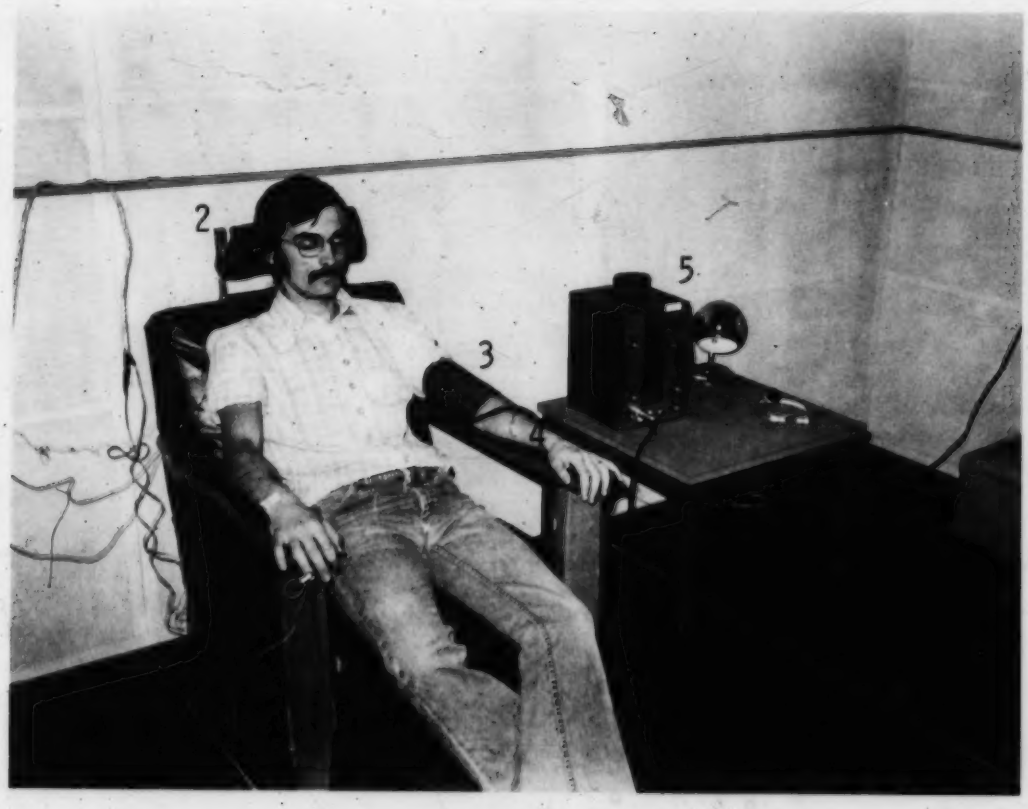

FIGURE 3. TESTING CHAMBER. This chamber is 9 x 12, sound and electrically isolated. Chamber is dimly lit during feedback sessions. #1. Digital plethysmograph; #2. Headrest, supporting head and securing O_2 electrode connection; #3. Standard blood pressure cuff; #4. Electrode for heart rate recording (LEAD I); #5. Avionics pressurometer, monitors blood pressure every 3.5 minutes; external speaker (not shown) from Narco Biofeedback Instrument situated 10 feet in front of subject; Intercom (not shown) placed on wall above and to side of subject.

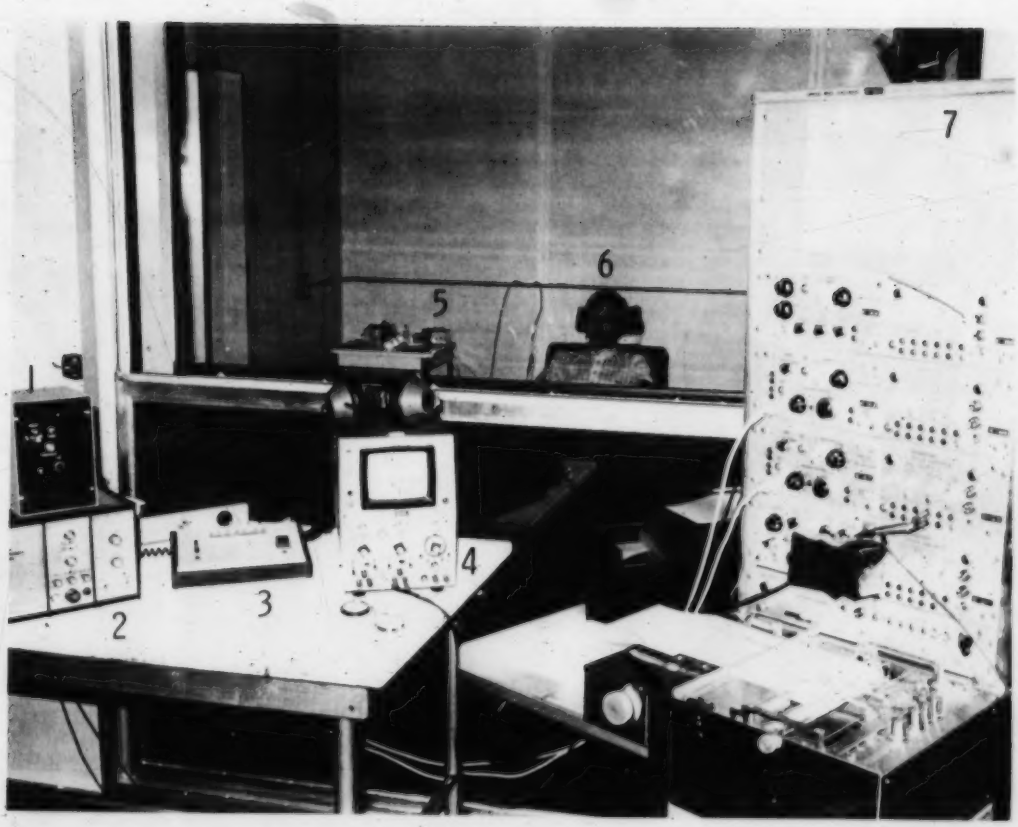

FIGURE 4. EXPERIMENTAL CHAMBER. This chamber is 9 x 12 and includes all monitoring instrumentation required. #1. Gated electronic timer; #2. Narco Biofeedback Instrument; #3. Intercom system; #4. Hewlett-Packard Oscilloscope; #5. Narco Preamplifier (time constant 0.3); #6. Subject as seen by experimenter through one-way mirror; #7. Grass Model 50 Polygraph: Channel 2 heart rate, channel 3 filtered EEG from Narco Feedback Instrument, channel 4, pulse pressure volume.

b. An explanation of the experimental procedure. The following procedure was used to increase the amount of alpha activity. An instrument is used to detect whenever alpha activity occurs. When alpha occurs a tone is emitted from a speaker situated 8 feet away. As the amount of alpha increases, the tone proportionally increases in frequency. Therefore, there is a two-fold task: firstly, to get a tone on and secondly to increase the sound of the tone (Travis, 1974, 1975). A number of initial suggestions were made; helping subjects produce alpha. Subjects were told that they may want to blank their mind or think pleasant thoughts. They may want to concentrate on their breathing or experiment with a variety of mental actions. Subjects were also told that they may notice an increase in saliva flow, fluttering of the eyelids, tingling in the fingers or a loss of sensory awareness. These were all indications of complete relaxation.

c. Signing of consent forms.

d. Answering the Taylor Manifest Anxiety Scale (Taylor, 1953).

e. Subjects were then shown the testing chamber and an explanation was made of how physiological activity would be monitored.

f. Finally, baseline (no feedback) measures of physiological activity were made. When the electrodes were attached the experimenter left the room and established contact (via intercom) with the subject. A two minute eyes open recording was made. The subject was then asked to close his eyes and relax. This ran for 15 minutes. Finally, there was a two minute eyes open recording made. The experimenter then returned to the testing chamber and removed the electrodes (approximately five minutes).

g. Finally, a subjective questionnaire was filled out by the subject.

The following words were used to describe subject's feelings at that moment. The appropriate number (1, 2, 3, 4, 5, 6, 7, 8, 9) was to be placed beside each word: interest, alert, annoyed, scared, discouraged, engaged in thought, enthusiastic, irritated, angry, anxious, drowsy, relaxed, tense, pleasant. Number 1 signifies slight agreement, and number 9 signifies strong agreement.

This questionnaire was used to evaluate motivation and interest throughout the duration of the program. It was also used to help each

subject try to identify quantifiably how they feel at the end of each session.

Subjects' expectations and impressions towards biofeedback has recently been shown to be important in determining the success or failure of conditioning alpha activity (Hart, 1968; Valle, 1975). All subjects were told to try and practice at home the techniques they had learned in the lab. They were instructed to sit down about the same time every day and try to go through their method of producing alpha activity (for approximately 15 minutes).

The remaining 13 conditioning sessions followed the following procedure (Table 3).

At the conclusion of Period 3 subjects were asked how they felt they did. Hypertensive subjects were asked to guess whether their BP dropped or rose and were then told what it was. Arrhythmia subjects were informed about their blood pressure and heart rate.

About one month after the conclusion of the experiment, the arrhythmia subjects were asked the following questions by their physician:

1. Has the frequency of occurance of fluttering or arrhythmia decreased any since biofeedback?
2. If frequency has not decreased, has severity?
3. Do you find yourself less anxious about the occurance of an attack?
4. Have you become more aware of how to relax mentally or physically?
5. Any comment?

TABLE 3

EXPERIMENT PROCEDURE FOR EEG ALPHA CONDITIONING. METHODOLOGY CONSISTENT FOR REMAINING 13 SESSIONS.

	PERIOD 1	PERIOD 2	PERIOD 3	
Electrode Attachment. 10 - 15 minutes. Subjects asked to guess blood pressure.	Prefeedback Recording. Eyes open. 2 minutes. 1 blood pressure measurement taken.	Feedback Recording. Eyes closed. Alpha conditioning. 15 minutes. 1 blood pressure reading every 3.5 minutes.	Postfeedback Recording. Eyes open. 2 minutes. 2 blood pressure readings.	Filling out subjective questionnaire.

Experimenter leaves chamber. Communication via intercom established with subject. Physiological equipment on.

Feedback on.

Feedback off.

End Session. Experimenter enters chamber. Removes electrode. Subject asked how he felt he did, then told heart rate and blood pressure.

Data Analysis

Determination of blood pressure was done by taking the average blood pressure for each individual period. This pressure was then converted to Mean Arterial Pressure by the following formula:

$\bar{P}a = Pd + 1/3(Ps - Pd)$, where $\bar{P}a$ = Mean Arterial Pressure
Pd = diastolic pressure
Ps - systolic pressure

(from Berne & Levy, 1972).

Percentage alpha time was calculated by taking the total time alpha recorded (from gated electronic clock) and dividing by the total time for that period (Period 1, two minutes; Period 2, 15 minutes; Period 3, two minutes). This was then multiplied by 100:

$$\frac{\text{Time from Electronic Clock}}{\text{Total Time for Respective Period}} \times 100 = \% \text{ alpha.}$$

It has been demonstrated that integrated alpha (i.e. the power under the waveforms) correlates about r = .85 with 30% to 70% alpha activity. This correlation decreases to about r = .45 at higher or lower percent time of alpha.

The Taylor Manifest Anxiety Score was marked out of 50 (N = 16).

The subjective questionnaire answered at the conclusion of each session was used to indicate changes in motivation and interest throughout the duration of the program. Objective data was also obtained by subtracting the evaluated number beside each word from the same word of the previous session; therefore, either a positive or negative change could result.

Approximately one month after the conclusion of the program,

arrhythmia patients were asked questions relevant to their specific disorder (given on page 42). These verbal responses were then tabulated.

RESULTS

The purpose of the experimental design was to observe any changes in patients' clinical symptoms, percentage alpha activity or whether any intra-or inter relationships existed between physiological and subjective data.

Change in Percentage Alpha and in Sessional Subjective Evaluations

The mean percent 'criterion alpha' emitted by each of the three groups (control, hypertensive, arrhythmia) is given in Table 4 (also graphically illustrated in Figures 6, 7, and 8).

The normotensive (control) and hypertensive groups showed increases in alpha activity for all three periods.

Controls:	Period 1; 5% - 55%
	Period 2; 69% - 84%
	Period 3; 13% - 59%
Hypertensives:	Period 1; 40% - 48%
	Period 2; 70% - 93%
	Period 3; 32% - 68%
Arrhythmia:	Period 1; 7% - 12%
	Period 2; 35% - 35%
	Period 3; 16% - 15%

TABLE 4

MEAN PERCENTAGE ALPHA ACTIVITY FOR THE CONTROL GROUP AND THE TWO EXPERIMENTAL GROUPS (HYPERTENSIVE AND ARRHYTHMIA)

	Session	1	2	3	4	5	6	7	8	9	10	11	12	13	14
	Period														
Normotensive Control Group	1	5	24	33	23	26	30	45	42	36	41	57	55	--	--
	2	69	76	88	80	81	83	82	83	81	80	83	84	--	--
	3	13	18	36	37	29	35	34	61	58	49	52	59	--	--
Hypertensive Group	1	40	28	57	55	53	57	60	68	65	53	53	55	47	48
	2	70	77	90	88	82	92	90	96	91	86	93	95	95	93
	3	32	27	39	37	37	63	62	67	73	62	78	75	57	68
Arrhythmia Group	1	7	8	9	20	19	23	17	9	4	1	4	3	4	12
	2	35	34	48	42	67	38	32	43	22	19	8	25	28	35
	3	16	20	23	38	40	55	16	45	15	3	4	3	20	15

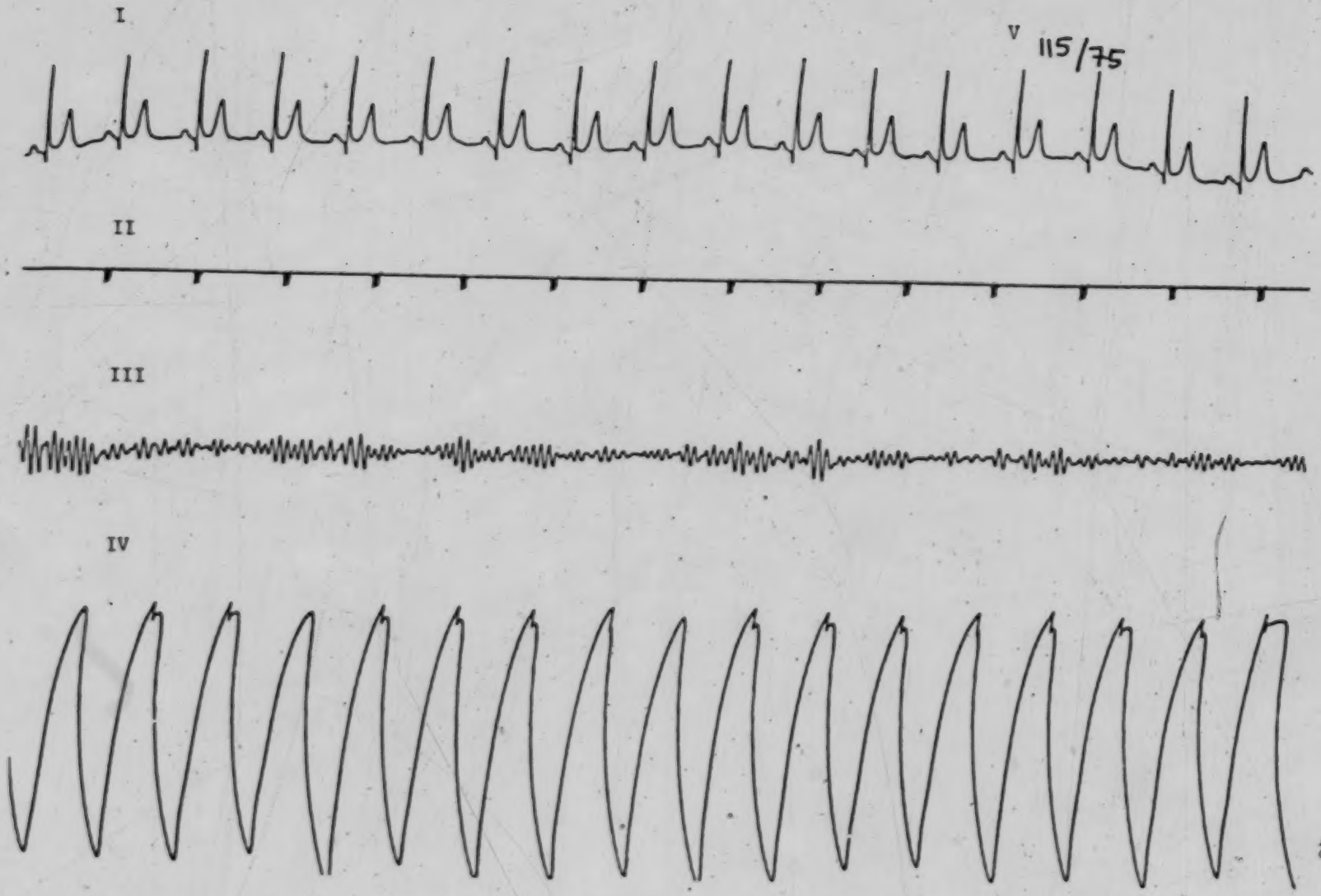

FIGURE 5. Typical strip chart recording paper speed 15 mm/sec. I: Heart rate - Lead I; II: Second timer; III: Filtered EEG activity from Narco Feedback Instrument 25 uv = 5 mm peak to peak; IV: Pulse pressure volume; V: Blood pressure from Avionics Pressurometer.

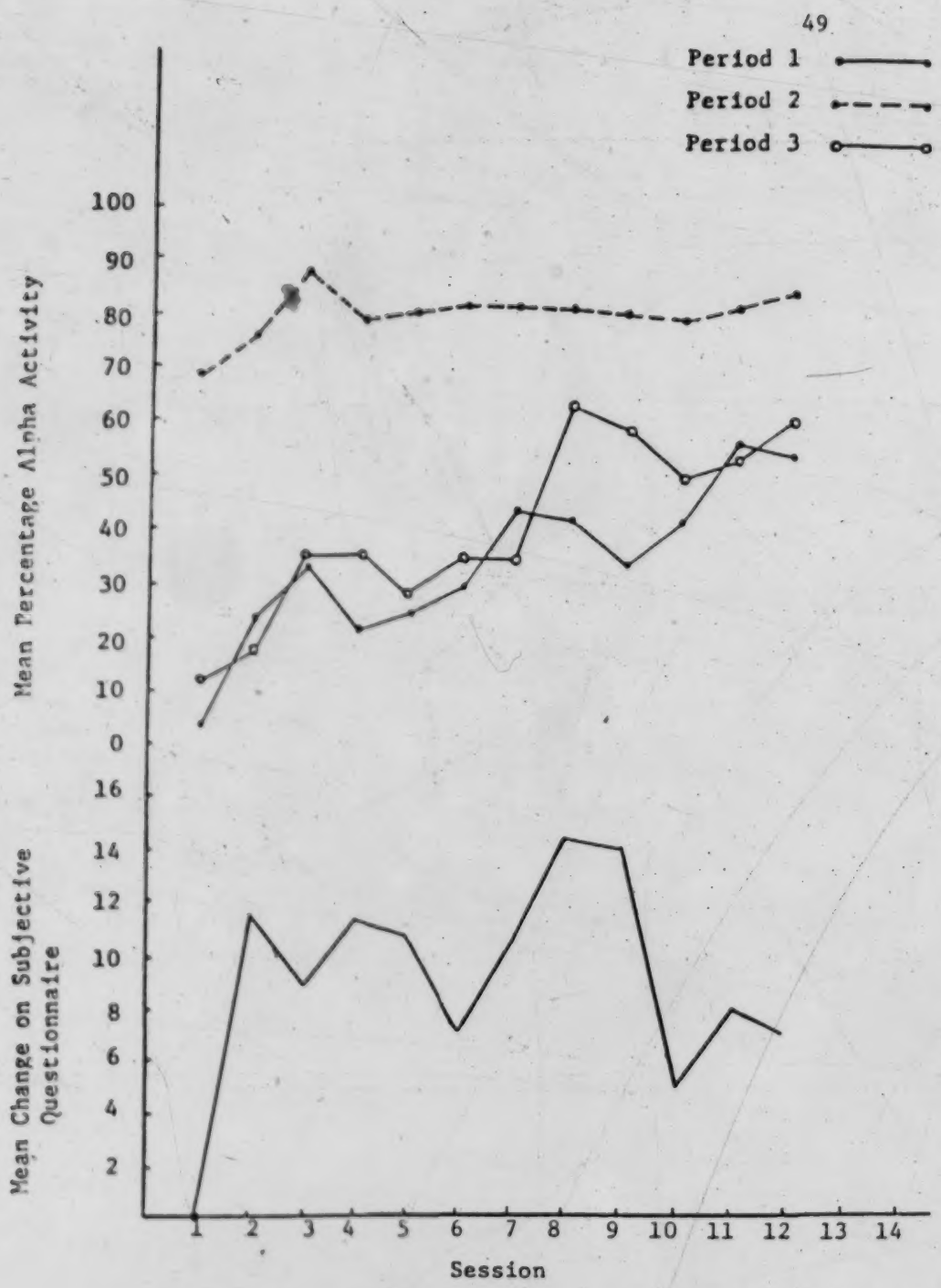

FIGURE 6. Control Group. The lower graph gives the mean changes on subjective questionnaires given at the end of each session. The upper graph illustrates mean percentage criterion alpha activity for each period.

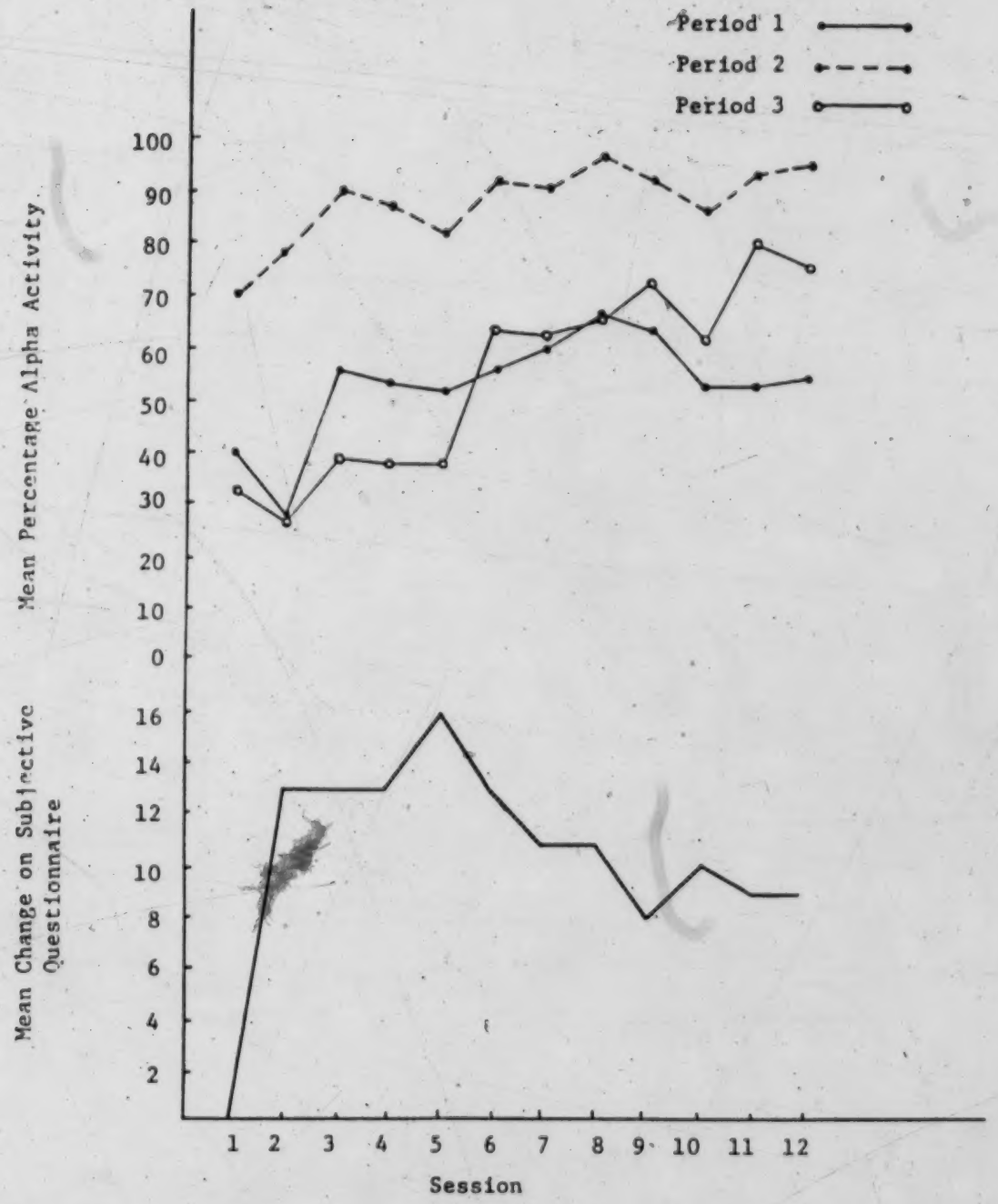

FIGURE 7. Hypertensive Group. The lower graph gives the mean changes on subjective questionnaires given at the end of each session. The upper graph illustrates mean percentage criterion alpha activity for each period.

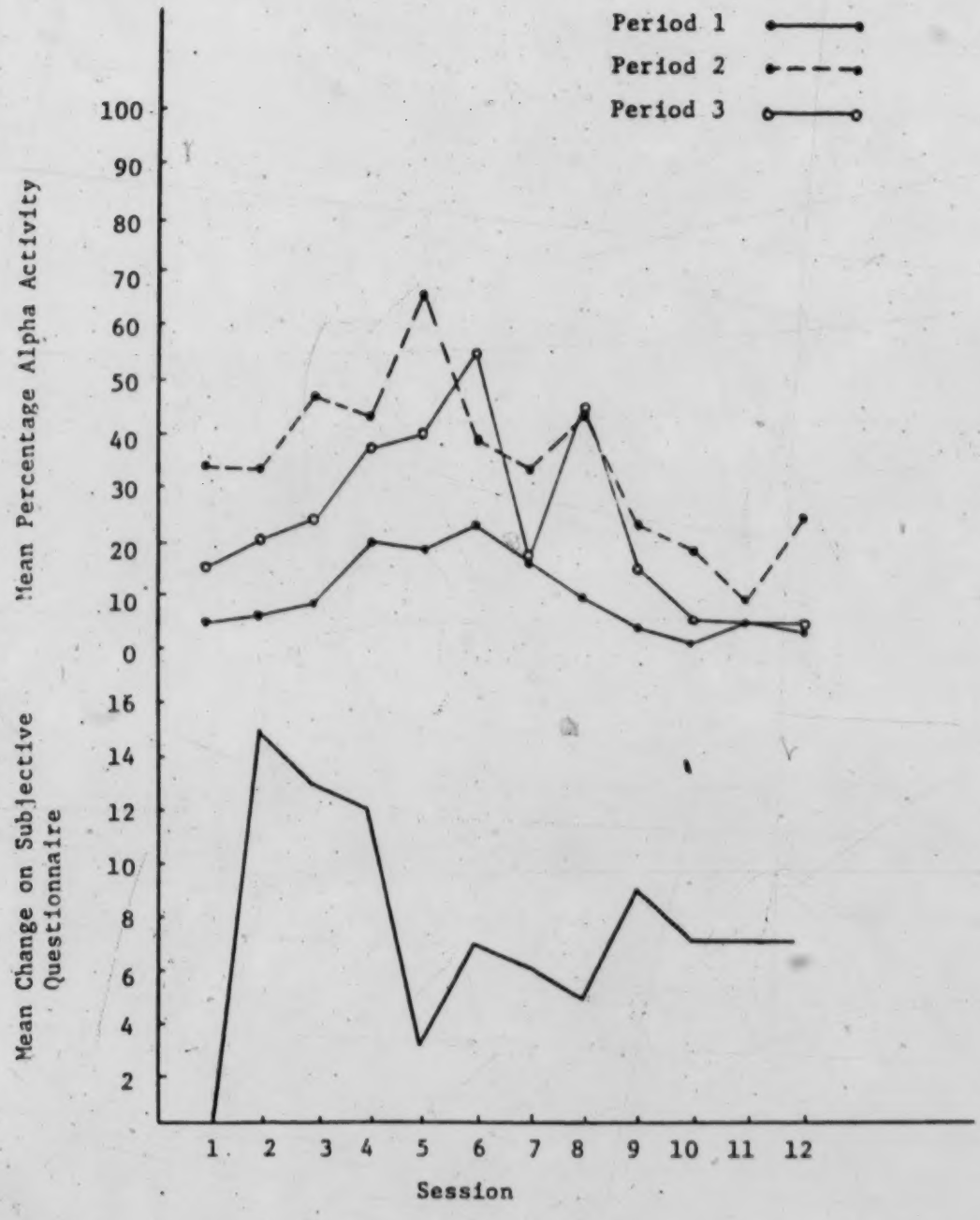

FIGURE 8. Arrhythmia Group. The lower graph gives the mean changes on subjective questionnaires given at the end of each session. The upper graph illustrates mean percentage criterion alpha activity for each period.

These increases tended to level off as the sessions progressed. The arrhythmia group followed a different pattern of change. There was an initial increase in alpha percentage (sessions 1-6) and then it began to decline, back towards the original baseline of session 1. This difference from the control group was statistically significant for all three periods (Period 1: $t = 4.6$; $df = 13$; $p < .001$; Period 2: $t = 1.02$; $df = 13$; $p < .400$; Period 3: $t = 5.7$; $df = 13$; $p < .001$).

The mean change in subjective evaluations for each group are given in Table 5.

The arrhythmia group registered the least amount of change per session (7.6); which is significantly different from the other two groups ($t = 2.81$; $df = 13$; $p < .025$).

Those words which reflected subject motivation and interest (interest, enthusiastic) remained constant (9 rating) for all subjects except hypertensive subject WL (progressive drop from 9 to 4 after session 7) and arrhythmia subject MP (progressive drop from 9 to 5 during sessions 9-14). When examining the therapeutic effectiveness of alpha training, we see these two individuals benefiting the least.

Relationships Between Percent Alpha and Other Physiological Parameters

Plotting mean percentage alpha activity and mean arterial pressure against the total number of sessions (Figures 9, 10, and 11), reveals a statistically significant inverse relationship (Period 1: $r = -.49$; $p < .10$; Period 2: $r = -.54$; $p < .05$; Period 3: $r = -.51$; $p < .05$).

TABLE 5

MEAN SUBJECTIVE EVALUATIONS FOR EACH OF THE THREE GROUPS.

THE MEAN SCORE ($\bar{X}$) PER SESSION IS ALSO INDICATED.

Group	Session	1	2	3	4	5	6	7	8	9	10	11	12	13	14	$\bar{X}$
Normo-tensive Control Group		0	12	9	12	11	7	11	15	14	5	8	7	--	--	9.2
Hyper-tensive Group		0	13	13	13	16	13	11	11	8	10	9	9	9	9	10.3
Arrhy-thmia Group		0	15	13	13	3	8	6	5	9	7	7	7	7	7	7.6

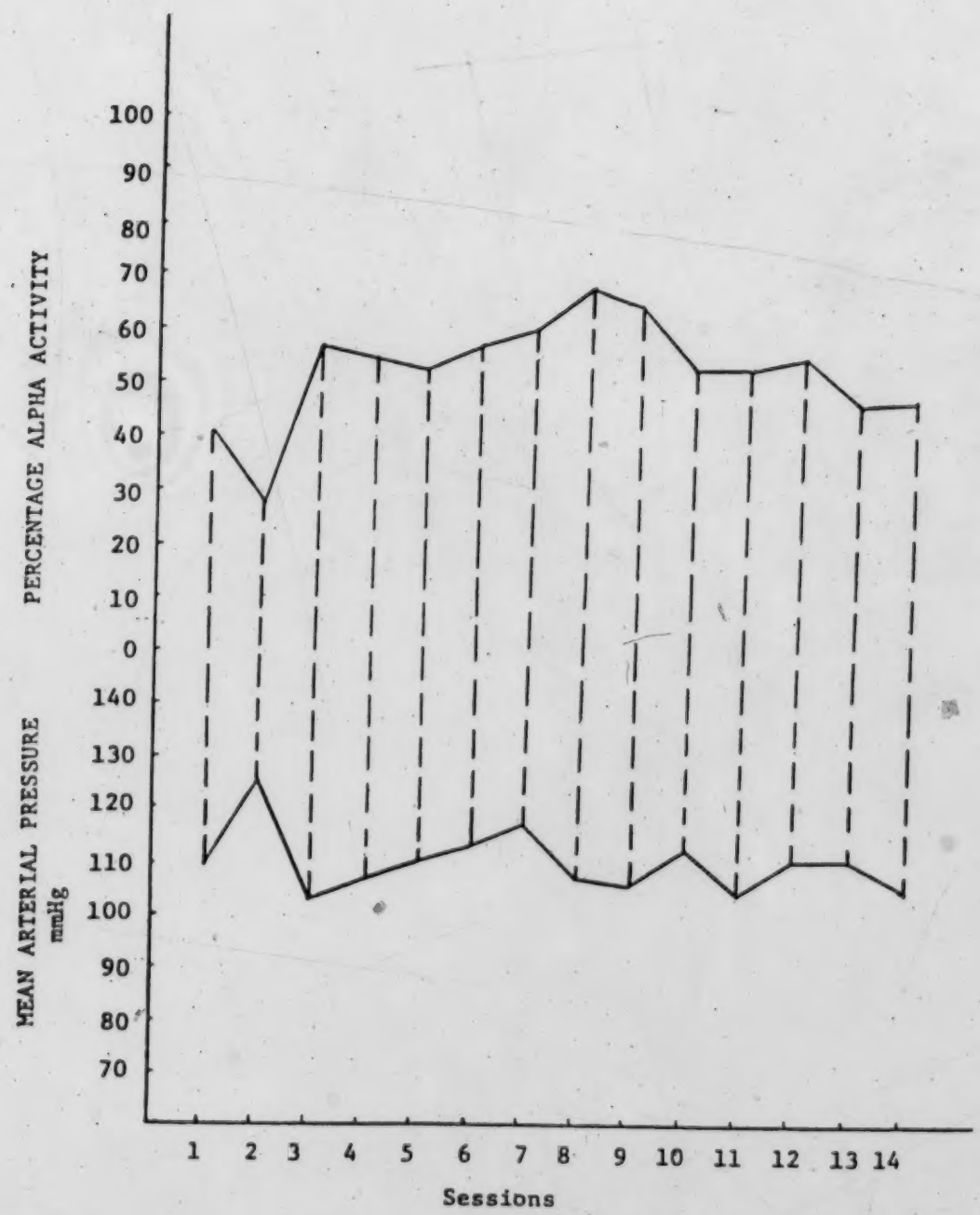

FIGURE 9. Period 1, eyes open, pre-feedback recording. Mean of the 3 hypertensive patients. Mean arterial pressure and percentage alpha activity vs total number of sessions.

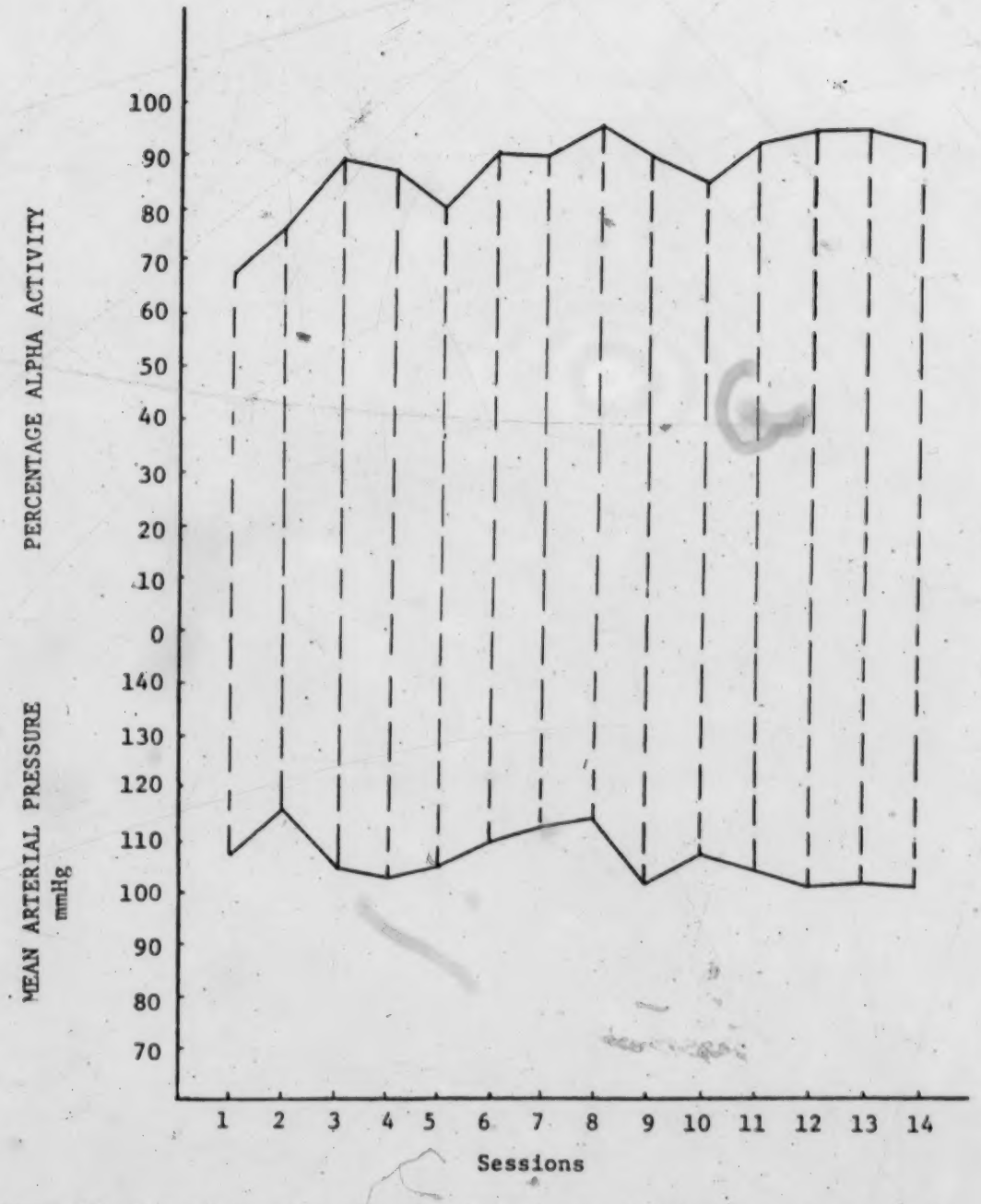

FIGURE 10. Period 2, eyes closed, alpha feedback sessions. Mean of the 3 hypertensive patients. Mean arterial pressure and percentage alpha activity vs total number of sessions.

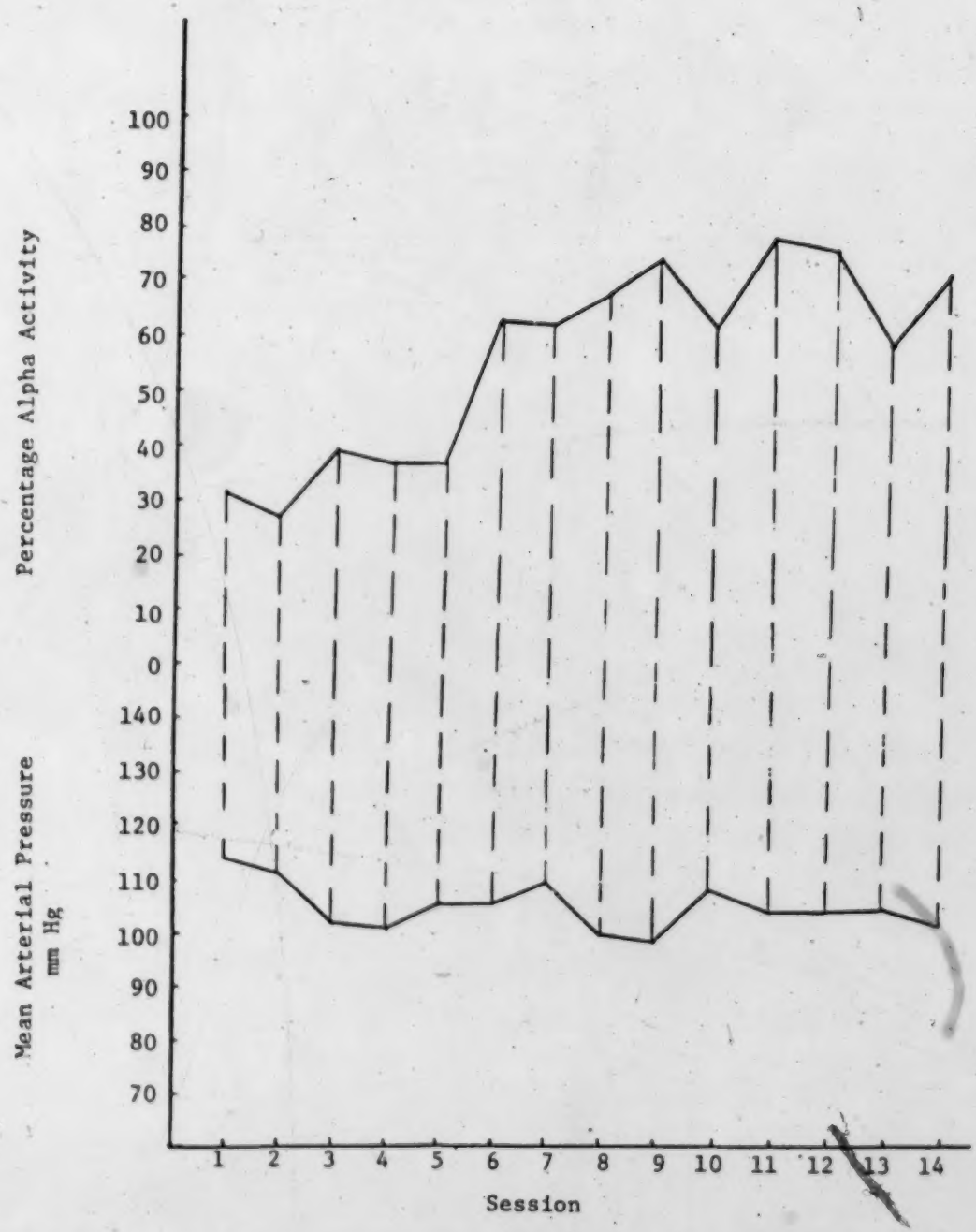

FIGURE 11. Period 3, eyes open, post-feedback recording. Mean of the three hypertensive patients. Mean arterial pressures and percentage alpha activity vs total number of sessions.

These correlations were found to exist for the hypertensive patients only.

No other associations between alpha and the other physiological parameters seem to prevail. Blood pressure and heart rate varied only slightly in the control and arrhythmia groups.

Pulse pressure volume was found to be an inconsistent measure and no quantifiable or qualifiable information could be extracted.

Changes in Clinical Symptoms Resulting from Alpha Training

The three hypertensive subjects show, in some cases, a statistically significant drop in blood pressure when comparing periods 1 and 2; 1 and 3, and 2 and 3.

TABLE 6

STATISTICAL CALCULATIONS FOR DECREASES IN BLOOD PRESSURE BETWEEN COMPARED PERIODS OF HYPERTENSIVES. PROBABILITIES ARE IN BRACKETS.

Subject	Periods Compared*		
	1-2	1-3	2-3
MM	.83 (<.500)	3.00 (<.010)	3.40 (<.005)
WL	1.30 (<.200)	2.20 (<.050)	2.60 (<.025)
EC	6.79 (<.001)	3.16 (<.010)	--

*Two-tailed t-test.

At the conclusion of the session (period 3), all three subjects showed a significant drop, from baseline, in their blood pressure.

Table 7 shows the difference in mean arterial pressure for each session, the average drop in pressure per session, and the average percentage drop per session.

In only one instance is there an increase in arterial pressure. Subject EC shows a slight increase (+2) from Period 2 (feedback) to

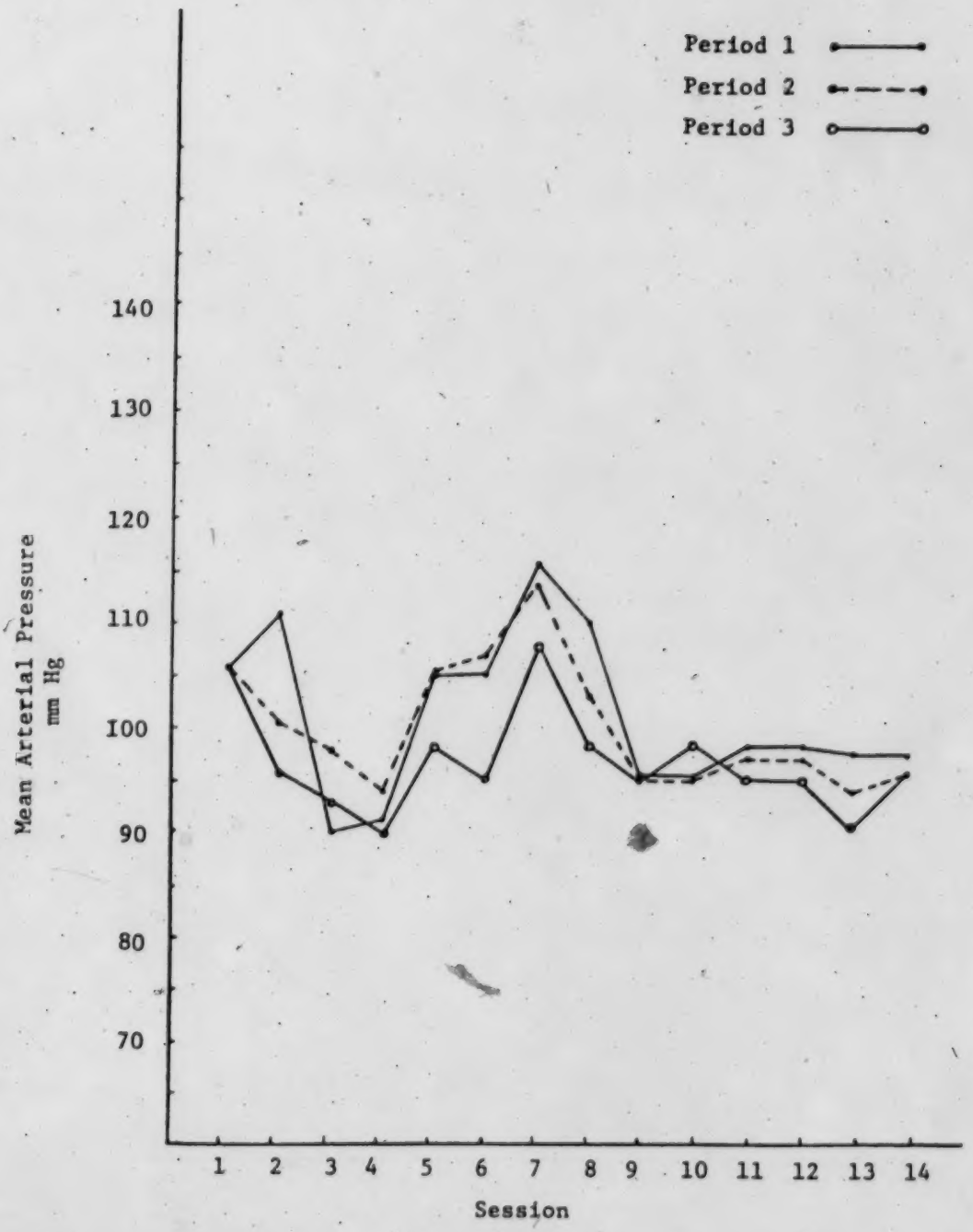

FIGURE 12. Hypertensive Subject MM. Mean arterial pressure for Period 1, Prefeedback; Period 2, Feedback; Period 3, Postfeedback.

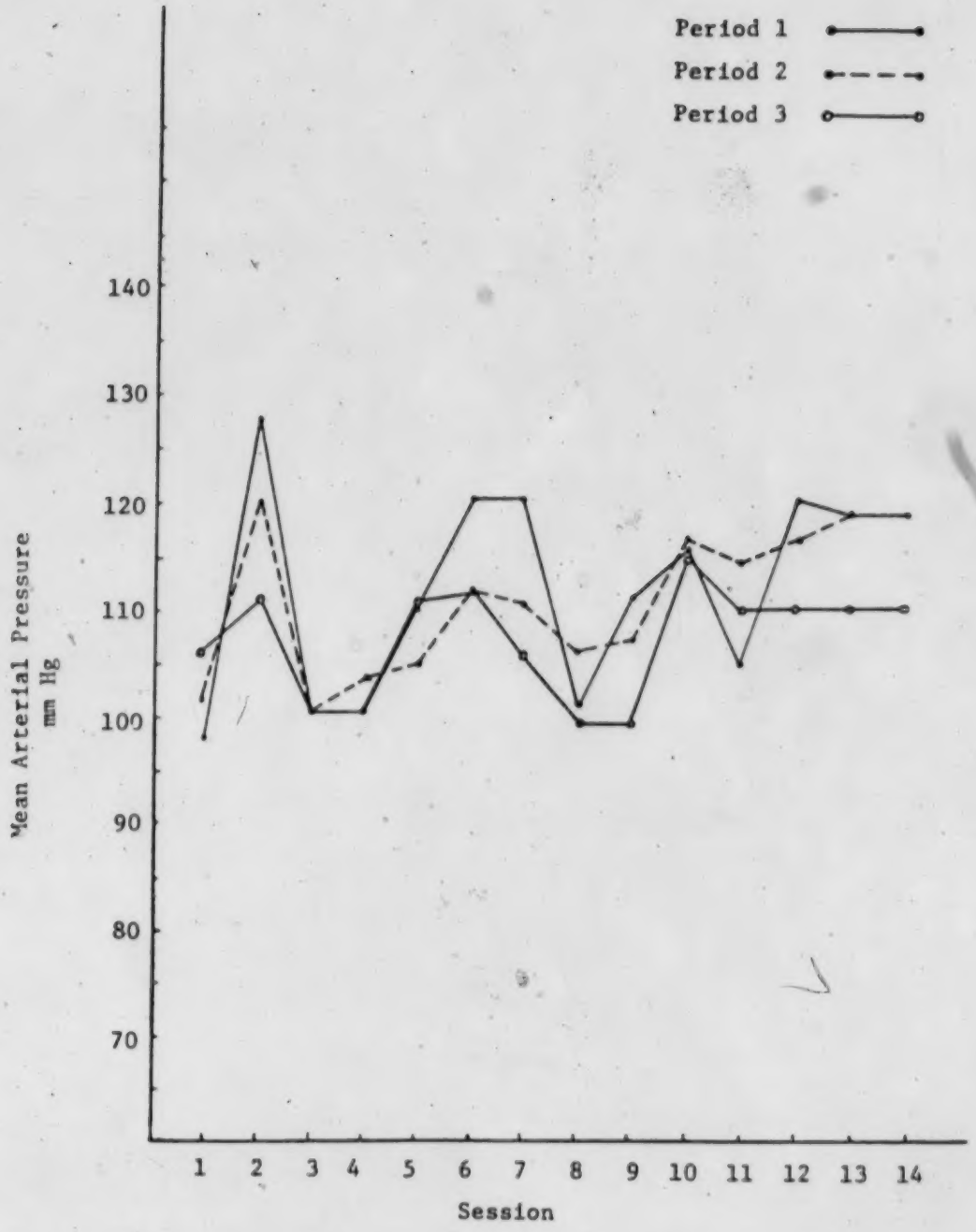

FIGURE 13. Hypertensive Subject WL. Mean arterial pressure for Period 1, Prefeedback; Period 2, Feedback; Period 3, Postfeedback.

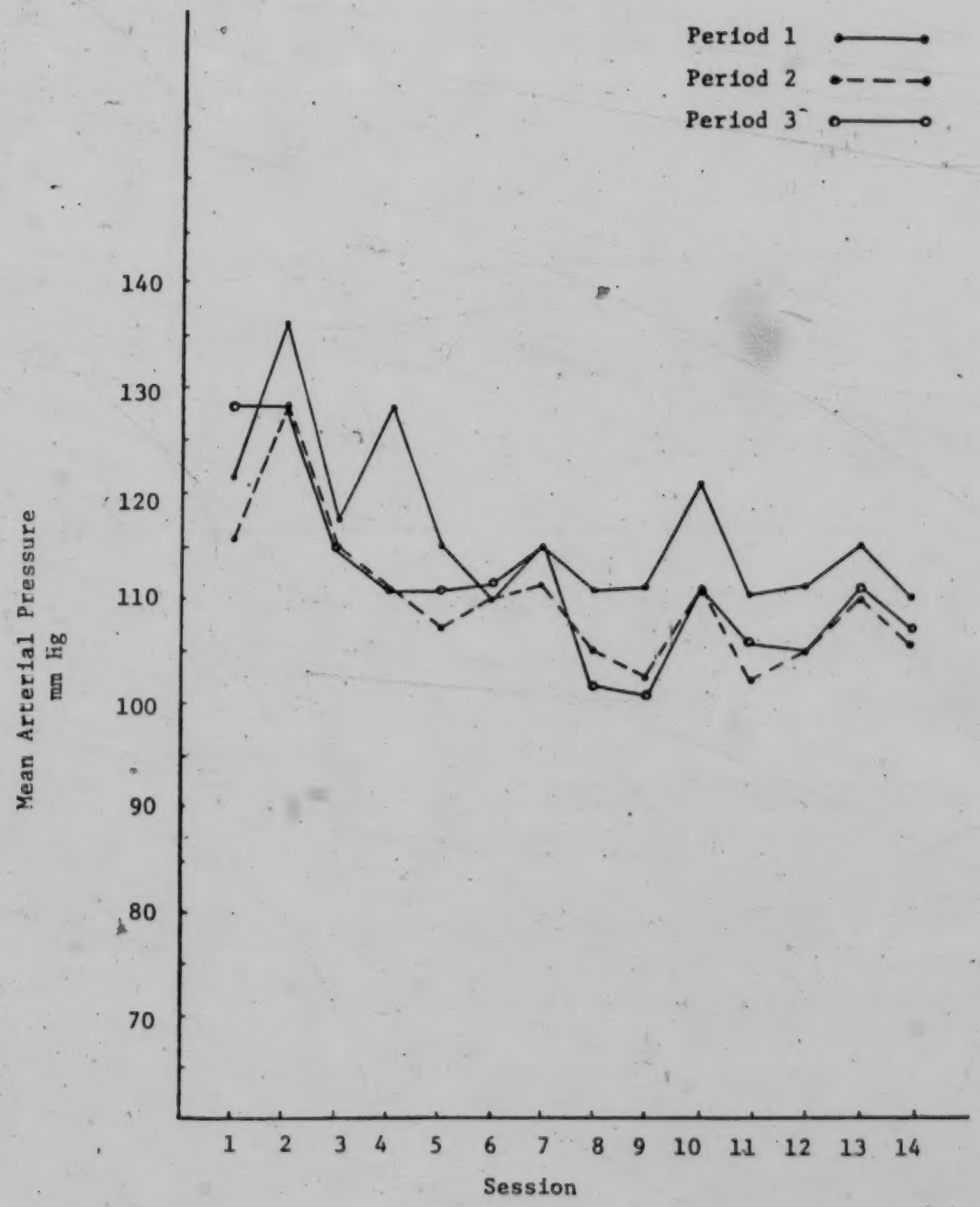

FIGURE 14. Hypertensive Subject EC. Mean arterial pressure for Period 1, Prefeedback; Period 2, Feedback; Period 3, Postfeedback.

TABLE 7

DIFFERENCES IN MEAN ARTERIAL PRESSURES BETWEEN PERIODS, FOR EACH HYPERTENSIVE. THE MEAN ($\bar{X}$) DROP IN PRESSURE PER SESSION AND THE PERCENTAGE CHANGE PER SESSION ARE ALSO GIVEN.

Subject	Periods Compared	Session 1	2	3	4	5	6	7	8	9	10	11	12	13	14	$\bar{X}$	Percent Change
MM	1 - 2	0	-10	+8	+1	+1	+2	-3	-6	0	0	-1	-1	-3	-1	-1.0	1
	1 - 3	0	-16	+3	-3	-7	-11	-9	-12	0	+3	-3	-3	-6	-1	-4.7	5
	2 - 3	0	-6	-5	-4	-8	-13	-6	-6	0	+3	-2	-2	-3	0	-3.8	4
WL	1 - 2	+5	-7	0	+2	-5	-9	-11	+4	-5	+1	+10	-6	0	0	-1.5	1
	1 - 3	+8	-15	0	0	+1	-9	-15	-5	-5	-2	+6	-10	-8	-8	-4.4	5
	2 - 3	+3	-8	0	-2	+6	0	-4	-9	-10	-3	-4	-4	-8	-8	-3.0	4
EC	1 - 2	-7	-8	-3	-15	-7	0	-2	-7	0	-11	-7	-7	-5	-6	-6.0	5
	1 - 3	+6	-8	-3	-15	-3	+3	0	-9	-11	-11	-3	-7	-3	-4	-4.9	4
	2 - 3	+13	0	0	0	+4	+3	+2	-2	-1	0	+4	0	+2	+2	+2.0	1

Period 3 (post-feedback).

Figures 12, 13, and 14 also show the session to session patterns of change in arterial pressure. When evaluating the overall therapeutic value of the program, subjects MM and EC show definite downward changes from baseline session 1. WL shows no such change; his blood pressure is very erratic from session to session, and this is especially evident in Feedback Period 2.

Because patient's medical records were available, a number of baseline measures were obtained. Table 8 tabulates the actual baseline blood pressures and compares it to the final blood pressure readings (Session 14).

MM and EC showed significant decreases in blood pressure from baseline measures to readings at the conclusion of alpha training program (MM: $t = 8.7$; $df = 2$; $p < .025$; EC: $t = 4.8$; $df = 2$; $p < .05$). MM had a 5% decrease in systolic pressure and an 18% decrease in diastolic pressure. EC showed 12% decrease systolic and 17% decrease diastolic. WL had little improvement; his blood pressure remaining clinically quite high. His largest drop was a 5% decrease systolic.

No objective measures could be extracted from the arrhythmia patients' data. It was important, therefore, to obtain complete subjective responses from the patients relating to their specific disorder. Table 9 gives their responses to relevant questions given one month after the conclusion of biofeedback training.

EM stated that she became less anxious about the occurance of an attack of tachycardia. She also stated that at home she could terminate heart flutter after about 2-3 minutes where before commencing feedback

TABLE 8

ACTUAL BLOOD PRESSURES AND MEAN ARTERIAL PRESSURES (BRACKETS) FOR HYPERTENSIVE PATIENTS. THREE BASELINE MEASUREMENTS AND THREE FINAL BLOOD PRESSURE READINGS (SESSION 14: PERIODS 1, 2, 3) ARE GIVEN.

Subject	MM	WL	EC
Last Recorded Blood Pressure by Physician.	134/100 (111)	180/104 (129)	143/120 (128)
Baseline Blood Pressure Reading, Session 1, Period 1.	140/90 (107)	135/80 (98)	165/100 (122)
Blood Pressure First Feedback Session 2, Period 1.	145/95 (112)	175/105 (132)	188/110 (136)
$\bar{X}$	140/95 (110)	163/96 (119)	165/110 (129)
Final Blood Pressure, Session 14, Period 1.	140/75 (97)	160/98 (119)	145/95 (112)
Final Blood Pressure, Session 14, Period 2.	130/80 (97)	160/98 (119)	145/87 (106)
Final Blood Pressure, Session 14, Period 3.	130/80 (96)	145/95 (112)	145/90 (107)
$\bar{X}$	133/78 (96)	155/97 (117)	145/91 (108)

TABLE 9

ARRHYTHMIA PATIENTS. RESPONSES TO QUESTIONS GIVEN ONE MONTH AFTER CONCLUSION OF BIOFEEDBACK PROGRAM.

Subjects	Has the frequency of occurance of fluttering or arrhythmia decreased any since biofeedback?	If frequency has not decreased, has severity?	Do you find yourself less anxious about the occurance of an attack?	Have you become more aware of how to relax? (mental or physical)	Any Comments?
EM	Doesn't think so.	Lasts only 2-3 minutes instead of 1/2 hour.	Definitely less anxious.	Yes. Both mentally and physically.	Feels it is a very good program and it definitely did help her.
MP	Doesn't think so.	No.	No.	Yes. Both mentally and physically.	She is very aware of her problem. Has cut out all tea and coffee. Uses very little alcohol. Went 8 months without an attack. Attacks are triggered by physical things such as the way she might move.
AP	Yes. She can alleviate an attack before it occurs. Fluttering can be conquered at home.	Definitely severity has decreased.	Less anxious.	Yes. Both mentally and physically very relaxing.	Breathing is very important. Has also helped in falling asleep. Less tired than previously.

training an attack would usually last 1/2 hour.

AP was able to prevent the occurance of tachycardia while at home. Her responses were similar to EM in that she felt less anxious about an attack occuring.

All three arrhythmia patients felt that they were more aware of how to relax mentally and physically. MP, however, when answering the relevant questions, felt that no change in her disorder occurred.

At the conclusion of the conditioning, there was no reduction in medication.

Subjective Comments During Alpha Conditioning

Both experimental and control groups expressed throughout the training that they became more aware of how they felt physically. If during one session their percentage alpha was slightly lower, they stated that physically they did not feel as relaxed. This was very interesting because generally subjects could not remember how well they did quantitatively on the previous session.

Towards the final sessions, hypertensive subjects were getting better at guessing their blood pressure even before the session began.

Arrhythmia patients ceased to mention that their fluttering was noticeable.

Hypertensive subject MM stated that he never felt better in all the years he had high blood pressure (18 years).

When asked how they controlled their alpha activity, "try to blank one's mind" was the most frequent comment. All subjects stated that producing alpha required some effort; it was not just spontaneous

upon closure of the eyes.

Although WL and MP seemed to lose interest (as evident by subjective questionnaires), no negative responses were made throughout the program. Generally when subjects did poorly in one session, they would seem remotivated for the next session.

When looking at trait anxiety (as determined by the Taylor Manifest Anxiety Score) and how it related to subjective evaluation, success of alpha conditioning or change in clinical symptoms, no relationships were found and this was probably a result of low subject numbers.

DISCUSSION

In discussing the results, a number of important questions are answered concerning alpha enhancement research, of which the most encouraging are those related to the therapeutic effectiveness of alpha conditioning.

Through multisessional alpha feedback training, subjects suffering from essential hypertension and those with functional cardiac arrhythmia showed changes in their respective clinical symptoms. The therapeutic value of alpha conditioning is illustrated by the fact that some of these patients showed favorable changes occuring in their respective disorder either within each session, by the end of session 14 (final feedback session), or both.

Generally, the observed responses by the hypertensives were in keeping with other documented findings; that through relaxation techniques, significant decreases in the elevated blood pressure occur towards a more homeostatic level (Deabler, 1973; Benson, 1974; Patel, 1975). Subjects MM and EC showed statistically significant drops in blood pressure (140/95 - 130/80 and 185/110 - 145/91, respectively) from baseline to session 14. These final blood pressures were also clinically normal. Although one subject (WL) showed no change in his clinical symptoms from baseline measures to session 14, his blood pressure did drop significantly within each session. This would indicate that one session did not influence another; however, the technique at the time was an effective therapeutic tool. Although subjects MM and EC showed a progressive downward change

in their blood pressure from session to session, a significant drop in blood pressure still resulted within each individual session. Blood pressures remained low after the feedback period indicating that the changes were not solely dependent upon the tonal feedback. Therefore, the tone seems to be used only as an indicator of how successful subjects were at relaxing.

Interestingly, MM showed less erratic changes in blood pressure from session 7; in fact, his blood pressure was not as labile. This is a significant result in itself as one of the defining characteristics of essential hypertension is that the blood pressure is labile.

Objective measures from the hypertensive subjects made accurate and consistent data a relatively easy task; especially when trying to evaluate changes in clinical symptoms. The arrhythmia patients, on the other hand, possessed no quantifiable measures. Even when subjects complained of heart fluttering during the sessions, no detectable changes in the ECG were observed. The problem with two of the arrhythmia subjects (EM and AP) was that this fluttering increased anxiety towards the occurance of a serious attack of tachycardia and eventually an attack occurred. The third subject (MP) stayed away from all stimulants (coffee; alcohol) and was also careful about certain physical actions (lying on her right side at night) for fear of initiating an attack. No detectable physiological reasons could be found for any of the attacks of tachycardia. The disease state solely seems to be related to the individual's psychic state. In order to evaluate the therapeutic effectiveness of alpha conditioning as accurately and consistently as possible, direct questions pertaining to their disorder

were asked. These questions were given one month after the conclusion of session 14, and by this time it was hoped that if any experimenter-subject interaction existed, it would have little effect now.

The results show a definite change in symptoms for two of the three patients. The two patients showing a lessening in the degree of anxiety about an attack. One patient (AP) was able to prevent the occurance of an attack while the other patient (EM) was able to limit heart flutter from 1/2 hour to 2-3 minutes. Subject MP indicated that there was no decrease in her anxiety about an attack. This was probably because she was convinced the attacks were due to physical stimulants only.

This decrease in anxiety was also evident in the hypertensive group. As the sessions progressed, subjects became more accurate at guessing their blood pressure. This sense of knowing how the blood pressure or heart reacts no doubt increased the chances of success of training.

WL and MP were the only subjects who showed a decrease in motivation and enthusiasm as feedback training proceeded. This supports previous findings that success in biofeedback training, and specifically autocontrol of alpha, was reflected by subjects' motivation and expectations (Hart, 1968; Valle, 1975).

Travis (1975) questions the effectiveness of alpha conditioning in chronic disease states. Subjects MM and AP had long histories of their respective disorder (18 and 40 years, respectively). It would therefore seem that the treatability of the disease (providing there was no organic cause) would depend on whether due to structural changes, a new homeostatic level had been attained, or whether the physiological

activity fluctuated periodically from normal ranges.

If alpha conditioning was effective for two very dissimilar disease states, then the question arises as to what extent can alpha training be applied therapeutically.

According to Dr. E. Green (1972) about 80% of all human ailments are psychosomatic in origin or at least have a psychosomatic component. Even if this estimate is high; the opportunities for applying alpha feedback training seem unlimited. Nideffer's (1972) question, relating to whether physical relaxation results from alpha conditioning, seems to have been answered. However, is this change a generalized relaxation response or does it pertain only to the autonomic nervous system and not somatic activity? Budzynski's (1972) finding of a small negative correlation between (frontalis) EMG activity and alpha occurance would therefore support a generalized relaxation concept.

Blanchard (1974) points out that to date the available data does not support the therapeutic application of biofeedback.

Relaxation and calmness seem to be general comments stated during most biofeedback training (outlined in the literature review). The question arises, is this subjective state a result of the direct control over physiological activity or does this state describe what subjects are actually 'learning' and as a result physiological changes occur?

Our results in conjunction with other findings may help in answering this question and how it relates to therapeutic success.

A great deal of success in treating essential hypertension has been gained through various relaxation techniques. As outlined in the literature review and again earlier in the discussion, these methods

involve meditation, hypnosis, yoga and GSR feedback training. Instead of controlling blood pressure directly (Schwartz, 1972), subjects are taught relaxation methods and hence to recognize relaxed states; as a result their blood pressure drops towards a more homeostatic level. Conditioning the EEG alpha rhythm resulted in mental clamness and physical relaxation and as a result blood pressure dropped towards a normal range and heart fluttering became less prevalent.

Although a great deal of documented research supports the conditionability of the autonomic nervous system, it would seem that greater therapeutic success is gained through attacking the root of the problem i.e., anxiety. The deciding factor in determining the therapeutic effectiveness of biofeedback may lie in the relationship between the etiology of the disease state (anxiety) and the concerned somatic target.

When applying biofeedback techniques to various disease states, the results in many cases uncovers more about the nature of the physiological system and their relation to cognition.

The findings of this study add further information about the characteristics of essential hypertension and cardiac arrhythmia.

A significant correlation between mean arterial pressure and alpha percentage does not necessarily mean one is dependent upon the other. If alpha conditioning results in physical relaxation then the relationship between the two is expected.

These findings, however, add little new information about alpha

and what it represents physiologically.

The present attitudes, as outlined in the literature review, support one of the two following views.

1. Alpha activity reflects inhibitory brain processes.
2. Alpha activity is an ongoing homeostatic rhythm occurring when those stimuli that normally attenuate the rhythm are blocked.

A recent article by Orne and Paskewitz (1975) would support statement 1. In their study shocks were given to subjects during alpha conditioning sessions. Even though subjects expected the shocks, they were still able to increase alpha density. This would suggest that subjects were actively inhibiting the reception of shocks, as reflected by the increased alpha activity.

Upon closure of the eyes, alpha activity occurs in 95% of the population. Therefore, it would seem that a decrease in sensory input allows ongoing alpha activity to occur.

Unfortunately, our results add little support to either view. Alpha may represent inhibitory brain processes suppressing those external (environmental) factors which normally attenuated alpha and eventually cause elevation of blood pressure. On the other hand, this blocking of sensory input may occur at another level (possibly subcortical) allowing alpha to occur.

The arrhythmia group shows completely different alpha conditioning than the normotensive or hypertensive groups. It is not immediately apparent why they show an initial increase in alpha activity and then a gradual decrease towards baseline. Stress and excitement influence

physiological activity along similar patterns. It is possible that initially alpha occurance was influenced by psychic states reflecting tension and anxiety. As the sessions progressed subjects became more aware of how to condition alpha. It is possible that at one point the enthusiasm was enough to affect physiological activity similar to that of stress. Subjects' enthusiasm remained high (physiologically suppressing alpha) and subjectively subjects still felt a sense of accomplishment thus deriving therapeutic value (see page 27).

Why this response is specific to the arrhythmia group is obscure; although it may answer a recent question brought up by Travis (1975):

> "Are there differences between (diagnostic) groups in success of alpha enhancement?"

This would therefore lead to the suggestion that the therapeutic value is not solely dependent upon the quantity of alpha production, but the recognition of the alpha state.

High alpha states certainly seem to reflect increased self-awareness This is supported by the fact that arrhythmia subjects scored less subjective change per session on their subjective questionnaires. The control and hypertensive groups seemed more perceptive in evaluating changes in mental and physical states. During high alpha states, subjects would be allowed the opportunity to become more aware of internal environmental changes instead of having to cope and interpret the constant bombardment of external stimuli. This would explain why hypertensives became more accurate at guessing their blood pressure and the arrhythmia patients became less anxious about fluttering and tachycardia.

Through discipline and biofeedback relaxation techniques, subjects

could become more familiar with conscious perception of their internal environment and thus learn to maintain a state of homeostasis at times of need.

Our results in conjunction with findings outlined in the literature review lead to a number of suggestions for future research.

1. Adequate control groups are necessary to rule out the placebo effect. It would be useless to run yoke controls within each session because; as illustrated earlier, especially with the hypertensive subjects, drops in blood pressure are maintained after the feedback session and from session to session. A more accurate approach would be to run a patient control group in conjunction with the experimental treatment group. Then, after the conclusion of the program, the control group can be conditioned. This method has recently been used by Patel (1975) and found to be effective. From an ethical standpoint, clinicians may question this procedure questioning whether it is proper to withhold treatment from patients in order order to establish placebo effects.

2. The greatest percentage increases in alpha were observed in periods 1 and 3. When high percentages of alpha were produced (period 2) little change was prevalent from session to session. It has been observed that strong coherences exist within the cortex in the production of alpha (Hord, 1972). Therefore; as we condition alpha, we not only see increases in percentage but also increases in the amount of regional activity (i.e. increase in voltage or amplitude).

For data analysis, integrated alpha would be a more accurate measure.

3. It would be useful to extend alpha feedback conditioning to other disease states and also monitor more organ systems (EMG, GSR, respiration) in an attempt to verify a generalized relaxation concept.

4. Map topographically movement of alpha during conditioning sessions. Hord (1972) did spectral analysis during alpha conditioning, however, he states only that alpha is the only rhythm that increases and secondly that strong coherences exist between frontal and occipital alpha.

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